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Othopedics

Functional Considerations in the Surgical Approach to the Hip-Joint

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One sometmes hears it said that arthoplasty of the hip-joint is a "precision" operation. This is not true and never can be true. It is a "salvage" operation. Nature's blueprint of the hip-joint is a masterpiece of construction and co-ordination. As a ball and socket joint, the hip permits of movement in three dimensions. The left and right hips must work together, yet be capable of controlled movement, mostly in opposite directions as may be beautifully seen in the evolutions of an accomplished figure skater. It must be capable of supporting immense weights (over 2 tons) and of taking the strain of transporting such a sportsman's trophy as the carcase of a bear weighing over 360 pounds over a distance of 5 miles.

While we are standing equally balanced on both feet, the mechanical equivalent is that of the loaded beam; when we are walking or running or going upstairs, the mechanical action is that of the balance. Whatever the activity, the function of the hip is always linked with that of the lumbar spine. The two form a complementary articulating apparatus. Under special circumstances the range of the two combined may far exceed the demands of ordinary life. It follows inevitably that no examination of the hip is complete without an examination, equally searching, of the lumbo-sacral region.

Let us review shortly some points in the anatomy of the joint, particularly in regard to the ligamentous and muscular control. The capsule of the joint is a massive sleeve which is attached inferiorly in front to the intertrochanteric line, and posteriorly to the neck of the femur. Its proximal attachment outlines the rim of the acetabulum. Part of this capsule is the strong Y-ligament, strong enough in itself to act as a check to hyperextension, and thus serve to economise muscle action.

The Ilio-Tibial Band

This may be thought of as an extra-articular ligament although it is usually described as a thickening of the fascia lata. It is much more than this. Inferiorly it has a wide attachment to all the bony points about the knee; from its deep surface there comes off the strong lateral intermuscular septum attached to the femur from the tendon of insertion of the gluteus maximus above to the lateral femoral condyle below. Traced in an upward direction, it gains attachment to the whole of the iliac crest by means of its continuity with the gluteal fascia, and by a deep offshoot it fuses with the lateral part of the capsule of the hip-joint. Its fibres run mainly in a longitudinal direction; it is controlled as regards tension by the tensor fasciae latae, and by fibres of the gluteus maximus.

The Muscles

Movement forward and backward is brought about chiefly by the powerful Ilio-Psoas and Gluteus Maximus respectively.

Adduction is provided for by a surprisingly large muscle mass. There are the three named adductors, and a transverse component is present in the piriformis, the pectineus, the obturator internus and the two gemelli the quadratus femoris and the obturator externus. It has to be remembered that the thrust of the body weight upon the head of the femur is not vertical but is inclined at an angle of about 165° to 170°. This involves a constant shearing force of variable intensity. In the absence of adequate transverse pull, the head of the femur must tend to travel outwards from the middle line of the body. It seems a reasonable suggestion that this outward displacement which so frequently accompanies degenerative changes in the hip-joint may be linked with weakness of the adductor component of the muscle control. One may even go further and speculate whether decreased blood supply consequent on injury or advancing years, may not play a considerable part in the development of Malum Coxae Senilis.

It would seem advisable to revise the ideas of muscle origin and insertion respectively that we have derived from the dissecting manuals. One may generalize as follows: When the lower limb hangs free, origin and insertion of the pelvi-femoral muscles can be regarded as corresponding to the text-book description; when the lower limb is fixed as in standing or walking, the functional origin of the homo-lateral muscles is the femoral attachment, the functional insertion is the pelvic. This holds for all muscles and includes the great adductor mass. The main purpose of the adductors is stabilization of the pelvis upon femur. The theoretical amount of the thrust of the femoral head against the acetabulum can be estimated mathematically to an approximate figure.

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The reverse of adduction is abduction. Strictly speaking this should signify carrying the lower limb away from the middle line of the body, a movement not much used in ordinary life. The functional equivalent is preventing the pelvis tilting towards the opposite side. The muscles presiding over abduction are three in number, the gluteus medius, the gluteus minimus, and the tensor fasciae latae. All three are supplied by the superior gluteal nerve. The part played by these three muscles along with the ilio-tibial band has been beautifully demonstrated by Inman. The gluteal pair act as an extensible spring controlling tilting of the pelvis to the contra-lateral side. It must follow from this that any surgical measure which interferes with the integrity of the gluteal spring will lead to a permanent diminution of the power of the individual to maintain the pelvis horizontal. This loss of power involves a limp so characteristic that it is referred to as a "gluteal" limp. This one consideration should be a strong deterrent to detachment of the gluteal muscles from the blade of the ilium. It is rather surprising that the mass of the abductors forms only about 15% of the total muscle mass about the hip. The explanation of this apparent discrepancy is found in the action of the ilio-tibial band controlled by the tensor fasciae latae. This extra-articular ligament acts as a check to pelvic tilt. Nature tends to be economical in the use of expensive tissue such as muscle, employing fibrous connective tissue wherever the latter is functionally adequate. The massive ilio-tibial band reduces very greatly the work that would have to be done by the gluteal spring, and forms a second line of defence for this muscle team against overstretching. It is a living guy-rope limiting movement of the pelvis on an antero-posterior axis just as the Y-ligament limits movement on a transverse axis. It must again follow that any surgical interference which menaces the integrity of the Tensor Fasciae Latae will affect unfavourably the control of the pelvis in relation to the head of the femur.

According to Inman, the minimum static pressure on the head of the femur is 2.4 to 2.6 times the body weight. There are two conditions in which the disposition of these compression (and other) stresses is upset; Congenital Dislocation of the Hip, and paralysis of the gluteal spring as after Poliomyelitis. Both these conditions may be present in a single individual. The outcome is Bilateral Coxa Valga.

With these and other similar physiological considerations in mind, what should be the guiding principles in the surgical approach to the hip?

1. Avoid interference with nerve supply of muscles. Make incisions, as far as possible, between, not through nerve territories.

2. Separate muscles; do not split them. Apart from nerve damage, muscle which is a highly organized and very vascular structure can be permanently and irreparably injured by haemorrhage and tearing.

3. The approach should be flexible enough to permit treatment of any operable lesion of the hip.

4. The exposure should afford a good view of the field of operation.

The Postero-Lateral Approach.

In the buttock, the gluteus maximus is supplied by the inferior gluteal nerve; the gluteus medius, the minimus, and the tensor fasciae by the superior gluteal nerve. The line of approach lies between these nerve territories. Perhaps the simplest concept is to think of the hip-joint as being concealed by an overlapping double-breasted covering. Above and behind there is the gluteus maximus; this layer partly overlies the anterior fold which consists of the medius and minimus. In order to displace the posterior fold, it is necessary to split the gluteal fascia along the upper anterior border of the muscle, and to mobilize a considerable part of its insertion into the fascia lata of the thigh. To do this with minimal blood loss, it is advisable to split the fascia lata first in a line from upper anterior border of the great trochanter downwards in the line of the fibres for 6 or 7 inches. By raising this fibrous flap it is usually possible to define the anterior border of the maximus muscle itself. Care is necessary to split the fascia alone. The muscle itself should not be split or torn if bleeding is to be avoided.

As the muscle flap is retracted backwards, the medius presents itself. Its posterior border is characteristically rolled. Behind it is the tendon of the piriformis, and distal to that tendon is the obturator internus with the two gemelli. Still further distal is the quadratus femoris. At the lower border of this muscle we come upon the adductor minimus, the name given to the uppermost fibres of the adductor magnus; and between these two, if the thigh be rotated inwards, the lesser trochanter presents itself, with the tendon of the ilio-psoas attached. The sciatic nerve is, as a rule, not seen, but is easily exposed if this be thought desirable.

Now let us turn to the supero-anterior covering of the joint. A finger can be placed behind the posterior border of gluteus medius, and by rotating the limb outwards, and following the minimus downwards, the anterior border of that muscle is delimited, and its insertion into the anterior face of the great trochanter is defined. A blunt instrument can be pushed under the tendon of the minimus to meet the finger behind the posterior border of the medius. The attachment of the medius and minimus to the great trochanter is severed leaving enough tissue to allow of firm

re-suture. The tensor fasciae is not touched so that this great component of the mechanism of pelvic stabilization is left uninjured. When, with a broad retractor, the medius and minimus are retracted upwards and forwards the hip-joint capsule is fully exposed, behind, in front, and above.

The incision in the capsule of the joint is made first in the line of the neck of the femur from acetabular rim above to upper part of anterior inter-trochanteric line below. One must get sufficient room to deliver the head of the femur without violence or tearing, so this incision is followed by two cuts, the upper following the rim of the acetabulum, the lower following the attachment of capsule to anterior inter-trochanteric line. By this proceeding the posterior part of the capsule is left intact thus preserving the main blood supply to the neck of the bone.

The head of the femur is coaxed out of its socket by flexion and outward rotation of the lower limb. By this manoeuvre a clear view of the acetabulum as well as of the head of the femur is obtained.

After the appropriate surgery of the hip joint has been performed the head is returned to the acetabulum, the capsular flap is re-sutured. The minimus and medius are folded back into their original positions and re-attached. The rest is routine.

In a few cases the great trochanter was chiselled across its base, and retracted upwards and forwards with muscle attachments intact. The method did not seem to have any advantages and was abandoned. Once or twice, instead of severing the muscle attachments, a thin shell of cortical bone was peeled from the outer surface of the trochanter. This procedure also appeared to be profitless, and was given up.

The postero-lateral approach described above is almost bloodless, almost shockless. It is extremely simple and can be made use of in any condition affecting the hip or the buttock. It is now being employed in the open reduction of Congenital Dislocation of the hip-joint, but perhaps that is another story.

Radiology

Ionizing Radiations and Cancer

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Roentgen discovered X-rays in 1895. In 1899 a cancer on the nose of a woman was treated by these rays and had not recurred 20 years later. During the same period of 20 years it is estimated that 100 radiologists died of radiation-induced malignant disease.

In 1896, Becquerel demonstrated the radioactive properties of uranium and by 1898, the Curies had separated polonium and radium. As in the case of X-rays, it was not long before radium had demonstrated its ability to produce changes in biological material and to produce, as well as to cure, cancer.

For many years X-rays and radium developed therapeutically along entirely separate lines, the former being employed by men who were primarily diagnostic radiologists, while the latter remained in the hands of surgeons. While, even today, it is not uncommon to find separate X-ray Therapy and Radium Departments, there has been a growing realization that the two agents, both of which act in the same way on the tissues of the body, can be used to best advantage by one who has made a special study of their properties. The result has been the emergence of a new specialist, the radiotherapist.

Today we are fortunate in that there has been rapid progress in the study of the nature of ionizing radiations, and of their effects on living cells, tissues, and organisms. In particular, we have amassed considerable knowledge of the conditions which favour the destruction of existing neoplasms rather than the production of new ones. The development of apparatus of increasing efficiency and power, though it has made the treatment of deep-seated tumors more simple and effective, has made the task of protecting the radiotherapist and his staff correspondingly more difficult. The problems of protection have recently been further complicated by the addition to the radiotherapists' armoury of a new weapon, the radioactive isotope. Physicists have long been working to evolve methods of measuring radiation in order that we may readily reproduce the conditions of treatment from patient to patient. The measure of their success in this science of dosimetry is that we may now speak confidently of the dose of radiation we are giving to the tumor itself, instead of quoting the "air dose" of X-rays or the "milligramme hours" of radium upon which we had so long to depend.

The Nature of Ionizing Radiations

Ionizing radiations are of two types:

1. Charged particles which directly produce ionization in their passage through tissues. Of these, the one in commonest clinical use is the beta particle or electron.

2. Electromagnetic rays which, during their passage through tissues, knock off electrons from atoms, thus producing indirect ionization. Both

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X-rays and the gamma rays of radium and cobalt 60 belong to this group.

The beta particle is limited in its power of penetrating tissues to a few millimetres. The penetrating power of X-rays depends on the voltage between the cathode and target of the machine that produces them. That of gamma rays depends on the energy released during the decay of the element which emits them and is constant for any element.

The Mode of Action of Ionizing Radiations

The biological effect of these radiations appears to be a result by the ionization they produce. Ionization entails the giving up of energy to the tissues. This is known to occur in several ways, depending on the type and energy of the radiation itself and on the composition of the tissues. In all cases, however, the final biological process is almost certainly biochemical in nature, probably affecting normal enzyme reactions by means of the products of ionization in the cells and in the tissues fluids which bathe the cells. At the levels of dosage used therapeutically, it is likely that the direct effect of radiation on the chromosomes is of less importance than these still imperfectly understood chemical reactions.

The Measurement of Ionizing Radiations

The ability to measure the dose of radiation which has been given to a patient is of prime importance. In the past, various empirical methods have been evolved to achieve this end. Examples are the "erythema dose" which measured radiation in terms of its ability to produce erythema in the skin. As this is a biological phenomenon and liable to great variation, it has not proved satisfactory. The change in colour of irradiated chemicals as in "Sabouraud pastilles" was also used for a time, but in the last 20 years the roentgen, a unit of dose of radiation based on the amount of ionization produced in air, has been universally adopted. It is worthy of note that today, as we employ radiations more and more diverse in type and energy, even the roentgen is proving inadequate owing to the failure of correspondence between biological and ionization effects where wide energy ranges and different types of particulate radiation are concerned. There is now a strong tendency to base dosage measurements on the most simple concept of all, the energy absorbed per gramme of tissue. The unit derived in this way will be known as the "rad."

The Irradiation of the Patient

Irradiation of the patient may be carried out in several ways, used singly or in combination.

1. External Irradiation.

The term external irradiation generally implies the use of X or gamma rays although beta particles, protons and neutrons are now being used in this way as well. The source of radiation is usually at a distance from the patient and th tumor may be treated through one or more field Generally speaking, the deeper the tumor and th higher its presumed resistance to radiation, th more complicated the set-up that is required. Now adays it is possible with a deep and radio-resistan tumor, to use some form of moving field therap commonly known as "rotation" therapy. In thi arrangement the tube is fixed so that the centra ray points at the tumor while the patient is rotate about his tumor. This results in a low dose t the skin and a very high dose to the tumor itsel

X-rays.

Depending on the voltage used to generate th X-rays, they are usually classified as follows:

a. Grenz rays, 10-50 Kilovolts, used for intra dermal and superficial carcinoma of skin.

b. Contact X-rays, 45-60 Kilovolts, useful i carcinoma of skin.

c. Low voltage X-rays, 60-150 Kilovolts, used i carcinoma of skin, lip, and superficial tissues.

d. High voltage X-rays up to 400 Kilovolts, fo carcinoma of breast and thyroid.

e. Super voltage X-rays above 500 Kilovolts for deep-seated growths. Rays in the first fou of these energy groups can be produced by norma X-ray machines. To produce super voltage X-ray it is usually necessary to use some type of electron accelerator such as a synchrotron, betatron o linear accelerator, or an electro-static generato such as the Van de Graaff machine. If desired X-rays can now be generated at energies up t one hundred million volts.

Gamma rays can be used for external irradia tion whenever a sufficiently large amount o radioactive material can be concentrated at on point. Two pieces of apparatus are in commo use:

a. Radium bomb, which contains 5-10 gramme of radium. It is difficult for various reasons among them that of cost, to use more than 1 grammes of radium and this limitation restricts the application of the unit to comparatively super ficial tumors, such as those of the head and neck

b. Cobalt 60 bomb. Such a unit may contai 1,000 to 3,000 curies of cobalt 60 and, when it is remembered that a curie of cobalt emits consider ably more radiation than a gramme of radium it can be seen how powerful these units are. Jus to emphasize the identity of gamma and X-rays the radiation from a cobalt bomb is equivalent t that from an X-ray generator working at abou 3½ million volts. Such units, of course, are use to treat the deepest growths, notably carcinom of the bladder, rectum, oesophagus, lung an brain.

2. Surface Application.

Radioactive surface applicators may be con structed to deliver either gamma or beta radiatio

to a superficial tumor. The gamma-emitting applicator is usually made of radium needles and tubes, radon seeds or cobalt 60 wire. A beta applicator can be made using radium by reducing the thickness of the metal screen. A "plaque" of this kind does not emit pure beta rays, as the gamma rays of radium are also present. On the other hand, artificial radioisotopes such as phosphorus 32 and strontium 90 emit pure beta rays and are to be preferred where, as in lesions of the cornea, there is an underlying structure, the lens, which it is desirable not to irradiate.

3. Implantation.

Implants of radioactive sources are used to treat accessible tumors by gamma radiation. They may be constructed of radium needles, radon seeds or artificial equivalents, such as cobalt 60, tantalum 182 and gold 198. Over the passage of the years, elaborate mathematical examination of the distribution of radiation around these implants has led to the formation of simple rules for their correct insertion.

4. Infiltration.

In some sites the direct injection of radioactive materials into the malignant tissues has given good results. The usual agent is gold 198 in colloidal suspension, which, used in this way, produces its effect almost entirely by its beta radiation. Malignant disease of the superficial tissues, the parametrium and the prostate have been treated in this way.

5. Intracavitary Irradiation.

A radioactive source, such as radium or cobalt 60 may be used inside a hollow viscus to irradiate the walls of the viscus. Instead of a solid source, a radioactive solution which fills the viscus may be used. Such a solution is normally held in a rubber balloon to prevent its absorption. Carcinoma of the cervix and of the corpus uteri, of the bladder and of the peritoneal cavity may be treated in this way.

6. Systemic.

Radium, given by intravenous injection, has been used to treat disseminated malignant disease, but the results were catastrophic because the fixation of the radium in bone and its long half life combined to produce further malignancy. Since the development of radioisotopes, with their short half lives and rapid elimination from the body, however, certain of these have proved of use, notably phosphorus in the blood dyscrasias and iodine 131 in carcinoma of the thyroid gland.

Radiotherapy and the Cancer Patient

Before commencing to treat a patient with cancer it is essential to decide whether the aim of treatment is curative or merely palliative.

In the first case, surgery and radiotherapy must not be considered as rivals, but rather as allies. Though there is usually a clear indication for one or the other, or, perhaps both in combination, in the treatment of a given lesion, the patient's interests are likely to be best served when the plan of attack is decided in consultation. Experience has shown that the overall improvement in results which follows this combined approach is very marked.

Where palliation only is possible, it is generally conceded that the burden falls on the radiotherapist. Whereas in the radical treatment of a lesion, certain reactions are to be expected and must be accepted by the patient, in the use of radiation for palliative purposes it is not justifiable to attempt to produce regression of a tumor at the cost of increasing the patient's misery. If this concept is adhered to, the radiotherapist can indeed help his patient not only be relieving the most trying of his symptoms, but by bringing him that mental solace, the realization that something is being done.

Obstetrics

A Three-Year Review of Stillbirths, 1951-53

At the Maternity Pavilion, Winnipeg General Hospital

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A review has been made of the causes of death of fetuses born dead at the Maternity Pavilion, Winnipeg General Hospital during the years 1951, 1952, 1953. There was no attempt at selection except that fetuses under the weight of 1,000 grams (2.2 lbs.) were not included in the overall analysis for the purposes of comparison with other centres. However, the uncorrected total figure of stillborns given in Table I includes all fetuses which were considered by the attending obstetrician to fall in the potentially viable class, i.e. were not abortions.

TABLE I
Winnipeg General Hospital, 1951-53

Total number of live births	10,870	
Total stillborns	189	
Stillborns over 1,000 grams	167	% live births 1.5
Total neonatal deaths	155	% live births 1.4 ∫ 2.9%

The review is based on the records supplied by the attending obstetrician, and the pathologist where a post mortem had been performed. In all, 89.7% of stillborns had autopsies performed. In many cases the records were inadequate for drawing any conclusion, either by the pathologist or for the purposes of this review. A plea is hereby made that all doctors record sufficient data on the hospital charts of any abnormal development of pregnancy, as it is only by continuous critical survey of our work that the present large fetal wastage can be materially reduced.

Bundesen reported recently a 14-year survey of 10,000 neonatal deaths in Chicago (all with satisfactory post mortem examinations and complete clinical investigations), which indicated that too many infants continue to die needlessly. 70% of early death following delivery was due to two causes — abnormal pulmonary ventilation and injuries at birth. More specifically, 19% of deaths in the first 3 days of life were due to intracranial hemorrhage; over half of these were premature babies, the majority of which died in the first day of life. The Chicago investigators found that the method of delivery was by far the most important etiological factor. This lays the responsibility of prevention of death directly on the obstetrician.

In making the present review of intra-uterine death only, it is realized that to make the picture complete, neonatal deaths should be included, as many fetuses born alive die within a few days of complications attributable to ante-natal or intra-natal conditions, i.e. poor maternal health, placental abnormalities or insufficiency, or difficult deliveries. However, the Pediatric department of the Winnipeg General Hospital has recently presented an analysis of neonatal deaths for the year 1953 and therefore this study is intended as a more detailed addition to the neonatal analysis.

When is a stillborn a stillborn? According to the regulations of the World Health Organization and adopted by the Province of Manitoba, any fetus born with a heart beat, but which never breathes, is considered a live birth. Formerly such a fetus was considered a stillborn, and in this analysis, two such cases came to light. On the hospital records they have been recorded as stillborn by the attending obstetrician, but in both instances there was indisputable evidence that the fetus had an audible heart beat and showed other signs of life although no obvious efforts at breathing were noted in the few minutes before death occurred. These cases have been eliminated from the records of intra-uterine death in this review.

Certain salient points are noted:

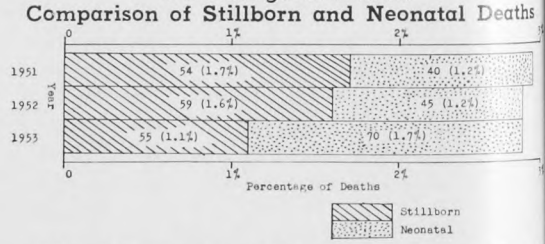
(1) Although in 1952 and 1953 (records are not available for 1951) the overall number of male births was almost identical with female births (1952—males 1,836, females 1,898; 1953—males 2,030, females 2,023), the proportion of males to females in intra-uterine deaths was 3 to 2 (Table V).

TABLE V
Comparison Sexes

	Male	Female
Premature	45	29
Mature	54	38
	99	67
No record	1	—

(2) As the percentage of intra-uterine death slowly falls during the years, the percentage of neonatal deaths increases, thus the total of the two remains fairly stationary. (Table II).

TABLE II
Percentage of Deaths
Comparison of Stillborn and Neonatal Deaths

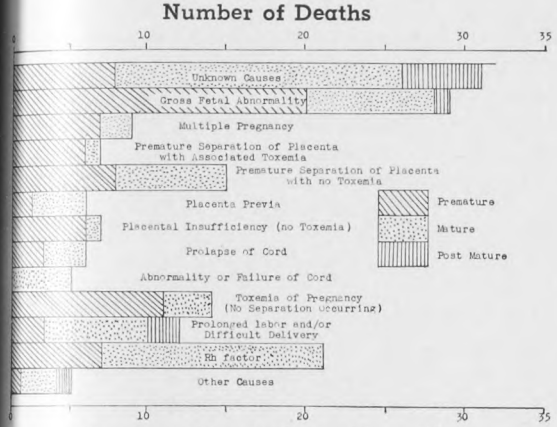


The decrease in stillbirth rate is due entirely to a drop in intrapartum deaths. (Table III).

TABLE III
Comparison
W.G.H. antepartum deaths to intrapartum deaths
(per 1,000 live births — weight over 1,000 grams)

	Total	Antepartum	Intrapartum
1951	17	9.0	8.0
1952	16	10.1	6.0
1953	11	8.9	2.1

(3) The Maternity Pavilion, having specialist staff members, attracts a greater number of maternity cases which may be considered complicated than would be admitted to hospitals with a smaller obstetrical service. This is particularly true of Rh sensitized women who are encouraged to come to the Maternity pavilion for the convenience of Dr. B. Chown and the personnel of his Rh laboratory. Thus erythroblastotic infants raise the stillbirth rate. (Table VI).



Causes of Death (Total No. 167)

(4) Contrary to popular opinion, the incidence of intra-uterine death of unknown cause in the so-called postmature patient (i.e. over 42 weeks), who is healthy and does not suffer from toxemia, is very low. In this analysis, comprising over 10,000 deliveries, there are only 2 cases in which there is no contributing factor; in both of these cases the data is incomplete.

There is one case that is apparently a true example of habitual intra-uterine death. The patient, who attends our prenatal clinics, lost 8 children at term either just before or during labor; only the fetus of her second pregnancy survived. The eighth pregnancy in 1951 was carefully followed during the prenatal period, labor was induced by surgical rupture of membranes at the 40th week only to have another infant die during labor. Accordingly when she returned pregnant again in 1953, an elective caesarean section was performed at 35 weeks and a normal living male child who has survived was delivered. It is pointed out that in this particular case pregnancy did not go beyond 40 weeks at any time to our knowledge.

(5) It is our experience that abruptio placentae, sufficient to cause death of the fetus, occurs without any evidence of toxemia (excluding histological comparisons of the placenta) approximately twice as frequently as with associated toxemia. The actual figures are: associated toxemia 7, no evidence toxemia 15.

(6) The incidence of intra-uterine death without apparent cause in the early portion of the third trimester remains high. Perhaps if there were some way of determining the cause of death despite the marked maceration that is invariably present, due to the delay of ensuing labor, a fair percentage of our stillborns might be salvaged.

(7) Although careful prenatal care, and care at delivery are given, our stillbirth death rate per 1,000 live births is almost double that of Chicago as reported by Potter. Chicago has the enviable distinction of having the lowest perinatal death rate of any large centre in the United States. We feel confident that the facilities at Chicago are no better than those in Winnipeg, therefore there is a challenge to every obstetrician in Winnipeg to do his part in attempting to reduce this incidence.

TABLE IV
Comparison
W.G.H. (1951-53) to other centres (per 1,000 live births)

		Total Still-births	Ante-partum	Intra-partum	Neonatal
Winnipeg Gen. Hosp.	(1951-53)	15.4	9.3	6.1	14.0
Kansas Med. Centre	(1949)	15.9	8.0	7.9	16.8
Chicago Lying — In	(1931-41)	18.9	10.7	8.2	17.4
	(1941-46)	12.6	7.9	4.7	12.6
	(1946-51)	8.8	5.9	2.9	10.8
New York Lying — In	(1952)	12.3			12.9
Middlesex	(1949)	27.2			30.6
Woolwich	(1951)	15.8			14.9
Dublin	(1951)	38.9			20.0

Stillbirth and Neonatal Survey

Following the preparation of this paper, a three-year survey of stillbirths and neonatal deaths at the Maternity Pavilion, Winnipeg General Hospital, and St. Boniface Hospital was begun. This survey has been authorized and financed by the Federal Department of Health and Maternal Welfare. All physicians using the facilities of these two hospitals are urged to co-operate in this endeavor by supplying all information pertinent to fetal deaths occurring in their practices to the appointed registrars in the two hospitals. It will be noted on reading the paper that a great number of cases could not be properly assessed due to inadequate information supplied by the attending physician.

I wish to acknowledge the assistance given me in the preparation of this paper by Dr. Elinor F. E. Black, Chairman, Department of Obstetrics and Gynecology, Winnipeg General Hospital. I am also indebted to the staff of the record department, Maternity Pavilion, for their co-operation.

Unknown Fetal Death, 1951

No Contributing Factor — 2 Cases

41 week female 3,700 grams. Death before labor. One normal living child. No cause of

death found in macerated fetus. Skin—yellow blebs. Rh factor not recorded. No toxemia.

40 week female 2,800 grams. Death during labor. No toxemia. 7 previous intra-uterine deaths. Only one living child. (Next pregnancy had Caesarean at 35 weeks with living normal fetus). (Staff patient).

Maternal Contributing Factor — 4 Cases

37 week male 2,930 grams. Staff patient. No prenatal care. Death before labor. Maternal anemia. Rh neg. P.M.: Not Rh factor but could be ABO reaction. Fetus too macerated to determine accurately. Two previous living babies.

39 week male 3,200 grams. First pregnancy. Two admissions to hospital for severe hyperemesis. Had perianal abscess post partum. No post mortem. Record inadequate.

35 week male 3,500 grams. Staff patient. First pregnancy. No prenatal care. Possible occult prolapse cord. Death during labor. P.M. Intra-uterine pneumonia. Not considered as the complete cause.

40 week female 2,500 grams. Staff patient. Death before labor. Maternal hgb. 37%. Pulmonary T.B. Also jumped through upstairs window 2½ weeks antepartum because house was on fire. Fetal death occurred about 10 days later.

Fetal Contributing Factor — 5 Cases

41 week male 3,450 grams. Private patient. One living child. External version done at 38 weeks. Fetal death one day before labor. Cord compression considered cause.

40 week male 3,450 grams. Private patient. One living child. Death due to asphyxia. Face presentation diagnosed. Patient given 100 mgm. demerol 20 minutes before birth. Death at completion second stage. No effort to breathe. (This death might be termed neo-natal).

40 week female 3,500 grams. Private patient. One previous pregnancy—living twins. Death before labor. Death might be due to anti-Kell reaction. (Dr. Chown).

40 week female 4,300 grams. Private patient. First pregnancy. Death ascribed to placental insufficiency. No post mortem nor pathologist's report on placenta.

40 week female 2,530 grams. Private patient. First pregnancy. Death during labor. Possible premature separation placenta. Record inadequate. Post mortem—asphyxia.

Unknown Fetal Death, 1952

No Contributing Factor — 2 Cases

39 week male 2,550 grams. Private patient. First pregnancy. Death before labor. No toxemia. Post mortem—macerated fetus. Record inadequate.

34 week female 1,750 grams. Private patient. First pregnancy. Death before labor. Mild toxemia. Attending doctor suggests abruptio as cause but inadequate record does not substantiate. No cause found in post mortem.

Maternal Contributing Factor — 3 Cases

41 week female 3,500 grams. Private patient. First pregnancy; two previous miscarriages. Death before labor. Patient had severe chill several days before labor. Post mortem—macerated fetus.

39 week female 2,550 grams. Private patient. Two living children. Virus pneumonia at 37 weeks pregnancy. Fetus died at height of infection. Post mortem—marked maceration. Death possibly due to maternal infection.

36 week male 2,100 grams. Staff patient. Eight previous pregnancies. (Second baby stillborn). One miscarriage. No prenatal care. Cord very short. Post mortem—macerated fetus. Death before labor.

Fetal Contributing Factor — 2 Cases

42 week female 3,000 grams. Private patient. 36 years old. Primipara. Mild toxemia. Patient had slight antepartum hemorrhage one day prior to labor. Following this fetal movement stopped. Post mortem—fetus macerated. No cause found, but death believed due to partial separation of the placenta.

37 week female 1,570 grams. Private patient. One living child. Death before labor. Macerated fetus but inter-ventricular septal defect found. (Note: same patient had an encephalic, 1953).

Cause of Death Unknown, 1953

No Contributing Factor — 5 Cases

35 week male 1,425 grams. Private patient. One living child. Death before labor. Post mortem—no cause found.

36 week male 1,250 grams. Private patient. Primipara. Death before labor. Post mortem—marked maceration.

42 week male 3,090 grams. Private patient. One miscarriage at 3 months. Death before labor. Post mortem—maceration.

42 week male 4,000 grams. Private patient. Primipara. Death before labor. Post mortem—no cause found.

32 week male 1,500 grams. Private patient. One living child. Death before labor. Post mortem—macerated.

Maternal Contributing Factor — 4 Cases

40 week female 4,400 grams. Staff patient. Six living children. 3 abortions. Patient obese, chronic bronchitis. Death during labor. Placenta was circumvallate. Post mortem—no cause found. Poor records.

34 week male 2,900 grams. Private patient. One set of living twins. Death before labor. Patient fell at 32 weeks. Low hgb. (49%). Post mortem showed interstitial acute pneumonia.

45 week male 3,500 grams. Private patient. One living child. Mother has multiple endocrine abnormalities, particularly hyperadrenalism. Death before labor. Post mortem — no adequate cause found.

36 week male 3,400 grams. Private patient. Three living children. Essential hypertension. Death before labor. Post mortem — antepartum intracranial hemorrhage (slight).

Fetal Contributing Factor — 4 Cases

40 week female 3,500 grams. Private patient. One living child. Death before labor. Death believed due to cord compression about neck. No post mortem. Poor records.

34 week female 2,300 grams. Private patient. Primipara. Death before labor. External version done 2 days before fetal movement stopped. Death due to anoxia. Possible cord compression. Also Rh negative with antibodies.

44 week male 4,545 grams. Staff patient. Four living children. Two miscarriages. Spinal anaesthetic 2 hours before birth. Admittedly too high. Death during labor. Possible placental separation. Post mortem—anoxia.

40 week male 3,500 grams. Private patient. One previous intra-uterine death. Cause not stated. Death before labor. Post mortem — asphyxia. Could possibly be due to hyalinization of placenta. (Poor records).

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Medicine

Portal Hypertension

J. H. Martin, M.D.

While hypertension of the greater circulation has been known at least since the days of Richard Bright (1836), and of the pulmonary circulation for many years, the term portal hypertension has been employed in medical literature for only about the past twenty years. The introduction of new methods of measurement of pressure and flow, along with the surgical approach to treatment, have added new interest to this subject.

The portal system is of great importance for at least two special reasons. Firstly, all water, nutrients, vitamins, minerals, drugs, toxins, etc., that are absorbed from the gut, pass through this system on their way to the liver. Secondly, a proportion of the oxygen requirement of the liver is supplied by venous blood. The exact proportion varies considerably in different animal species. The portal vein is unique in that it begins in capillaries which become venules, and ends in venules which become capillary in size. The pressure in the vein depends on the rate of blood inflow, the rate of blood outflow, and the "tonus" in the vein wall. Dale has described an hepatic sphincter present in the hepatic vein of animals and has reported its varying sensitivity to different drugs. This sphincter is probably important in the mechanics of shock in certain animals, but in the human it is probably non-existent or very weak.

It is difficult to state exactly just what is normal portal vein pressure. There are many variables such as the method of measurement, the systemic blood pressure, the depth of anaesthesia and the position of the patient. Macpherson, who has had considerable experience with measurement, states that the upper limit of normal in man under anaesthesia, with the abdomen open is 150 mm. of water. This is compared with the usual pressure of 50-120 mm. of water that exists in peripheral veins at heart level.

Etiology: Portal hypertension is probably never primary or essential but is always secondary to other factors. Extremely rare arterio-venous fistulae have been reported. The usual causes might be divided into three groups:

I. Supra-hepatic. This group would include: (a) Diseases of the heart and lungs especially with right sided heart failure. Since these patients rarely have their abdomens opened, we have no measurement of value in this condition. However, while ascites is very common, varices do not occur in this group. (b) Hepatic vein thrombosis (Chiari's disease), a primary obliterative endophlebitis, is very rare.

II. Intra-hepatic. This type is due almost entirely to some form of liver cirrhosis and the group is responsible for 85-90% of all instances of portal hypertension. Hypertension exists even in acute hepatitis, but probably has to be present for some period of time before it is evidenced clinically. Obstruction of portal circulation is usually considered to be due to fibrosis following repeated episodes of zonal necrosis. However, the degree of hypertension has often been observed as being out of all proportion to the amount of fibrosis. Kelty et al by use of plastic moulds have demonstrated convincingly that it is the regenerating hepatic lobules which bulge into the venules, distort the architecture, and obstruct portal flow. So there may be gross impairment of liver function without production of hypertension, or there may be advanced portal hypertension with maintenance of good liver function. A list of etiological agents producing cirrhosis would be out of place here.

III. Post-hepatic. Portal or splenic vein thrombosis occurring acutely produces hypertension and gastro-intestinal hemorrhage with rapid termination. Of the chronic group, Linton's classification is as follows:

1. Congenital
   a. Extension of the process which normally obliterates the umbilical veins into the portal system.
   b. Cavernous transformation, the exact mechanism is not clear but may be:
      i. a congenital anomaly.
      ii. an attempt at revascularization of the process in (a).
2. Acquired
   a. Thrombosis: infection, trauma, spontaneous.
   b. Extraluminal pressure: tumors, abcess, etc.

In hypertension, attempts at collateral circulation are established between the portal and systemic circulations. The sites at which these take place are well known. I would only like to point out, as shown by Anderson, that the greatest amount of portal-to-systemic flow takes place in the posterior abdominal parietes.

Clinical Features: The necessary length of time which hypertension must exist before it becomes evidenced clinically is variable but it is at least several months.

1. Hemorrhoids—one can only say again, as has been said so often, that this common and simple disorder may be a sign of serious or fatal disease.

2. Caput Medusae. This has aptly been described as existing far more often in text-books than in patients. Its mechanism is still controversial but it is probably the result of re-opening of obliterated umbilical veins.

3. Massive Hemorrhage. Due to ruptured esophageal varices, this condition has a high mortality, approaching 70% in many series. The mortality is higher when the cause is intra-hepatic. All the factors in production are not clear, but an important cause is peptic erosion by regurgitated gastric juice. Some hold that such erosion does not occur in the absence of hydrochloric acid in the stomach. Chiles, writing from the Mayo Clinic, surveyed material obtained over 30 years. He found ulceration to be present in about 60% of cases. The early diagnosis is difficult. The presence of an enlarged liver, or of spider angiomata, or the finding of bromsulphalein retention is an aid. Occasionally massive hemorrhage from varices may occur without giving any clue as to its source of origin.

4. Hypersplenism. Hypertension produces passive congestion of the spleen followed by fibrotic and regenerative changes with a resulting effect on peripheral blood. A pancytopenia may be produced, or any individual type of cell may be depressed singly. Whether this is due to increased phagocytosis or to humoral depression is still a source of argument. The presence of a normal or hyperplastic bone marrow distinguishes this from other like dyscrasias in most instances.

5. Ascites. This clinical sign is included for the purpose of pointing out that it is not the result of portal hypertension. There are probably several factors operative in its production including increased venous pressure, hypoprotinemia, variation in protein composition, un-metabolised anti-diuretic hormone, and hypersecretion of adrenal corticoids. However, raised venous and capillary pressure is not, in itself, enough to produce ascites. Therefore, treatment directed to the hypertension alone will not necessarily relieve the ascites.

Treatment. As this syndrome is the result of several diseases treatment is necessarily directed towards the primary cause. Most important is the treatment of hemorrhage:

A. Acute phase. This consists of:
   1. Transfusions — must be massive and adequate.
   2. Antacids — to neutralize gastric acidity.
   3. Patton bag — a useful method to apply direct tamponade to the lower esophagus.
   4. Thoracotomy with venous suture.
   5. Topical thrombin or gelofoam is being abandoned.

B. Prevention of recurrence:
   1. Various types of omentopexy, splenic artery ligation and hepatic artery ligation (Reinhoff) have been largely abandoned.
   2. Gastric resection to produce anacidity is recommended by Wagensteen.
   3. Shunting operation.

Shunting operations appear to be the most logical method of approach and have produced the most satisfactory results. Jahnke and his associates have recently reported on 30 cases. Whenever possible, they used a direct end-to-side portocaval shunt. Severe disturbance of liver function was not considered a contraindication. Following operation, the underlying liver disease was neither improved nor made worse. This is of special interest in view of the large amount of oxygen obtained by the liver of man from the portal blood. The mortality was only two cases. Recurrence of hemorrhage has been minor in two cases.

Progress is constantly being made in our understanding of the structure of the liver and especially of blood flow through the portal system (Elias). With this it is hoped will come new methods of treatment for liver disease and of the subsequent portal hypertension.

Physiology

The Clinical Significance of the Electroencephalogram in the Adult

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The electroencephalogram (EEG.) is a record of the minute electrical voltages produced by the matter of the cerebral hemispheres. The voltages are either picked up between pairs of electrodes placed on different areas of the scalp or between an electrode on the scalp and an electrically inactive or average electrode. The voltages across the electrodes are amplified electronically and their variations recorded by ink-writing pens on a wide strip of moving paper. Activity from many different areas may be recorded simultaneously on six to eight different pens.

For the test the patient sits in a chair or lies on a bed, thirteen to eighteen electrodes are placed over the frontal, temporal, parietal and occipital areas and the electrodes connected by flexible wires through switches to the appropriate amplifiers and recording pens. During the recording it is necessary for the patient to be relaxed to avoid obscuring the record with activity from the muscles of the head. Throughout the recording the eyes are opened and closed at frequent intervals since visual stimuli can significantly modify the activity from the brain. Over-breathing is performed to lower the blood CO_2, cause constriction of the cerebral blood vessels and so bring out any abnormalities that may be present. Photic stimulation with repetitive flashes from a high intensity strobe light is performed in order to evoke activity in the visual pathways, to test their functioning and on occasion to activate latent abnormalities.

To obtain additional information to that of the routine examination, the EEG. may be taken during activation by different physiological and pharmacological stimuli. The forms of activation currently used are sleep, barbiturates, insulin, glucose, metrazol, and triggered photic stimulation. Activity from the basal regions of the brain may be recorded from an electrode placed up the nose to lie against the sphenoid sinus.

The Normal Electroencephalogram

The normal EEG. shows the following basic characteristics: in the pre-central areas a low voltage fast activity is present, in the post-central, temporal, parietal and occipital areas a rhythmic pattern called the alpha rhythm appears when the eyes are closed. This rhythm has a frequency in the range of 8 to 12 cycles a second (c/s.), on opening the eyes the rhythm diminishes or disappears. Some persons show no alpha activity normally and absence of this rhythm is not necessarily abnormal. Some 50% of normal individuals show a little rhythmic slow 5 to 7 c/s. activity in the temporal areas. A typical normal EEG. tracing is shown in figure 1 but there are many variations of this example.

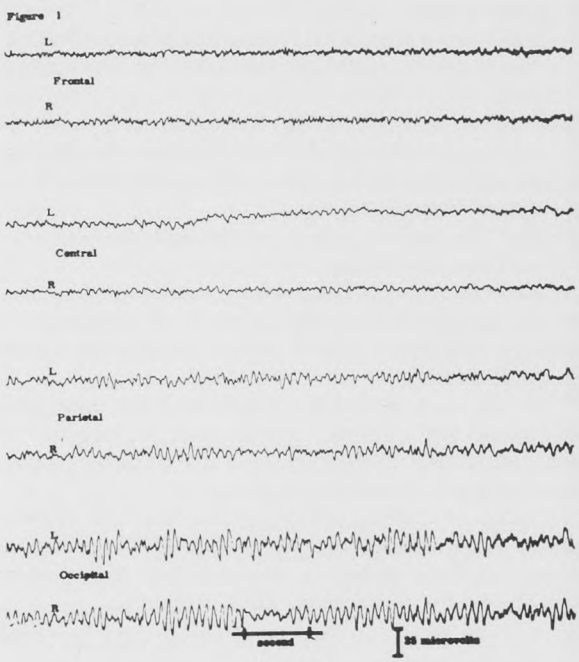

Figure 1
An example of a normal EEG. record. Recording electrode referred to Offner average electrode.

The presence of patterns other than those described above constitute abnormality in all but the elderly and the young.

The criteria of normality have been established empirically by taking EEG. tracings from large groups of physically and psychiatrically normal persons. The incidence of normal EEGs. amongst a normal group varies from 95 to 85%, depending on the group selected and the rigidity of application of the EEG. criteria. With the criteria used in the Manitoba centres some 93 to 95% of the adult population showing no evident neurological or psychiatric disorder might be expected to have normal EEG. records.

A Normal EEG. in the Presence of Known Disease

Although a normal EEG. suggests the strong possibility that no lesion is present affecting the cerebral hemispheres, from 10 to 15% of patients known to have a cerebral tumour[1] and from 25 to 30% of patients known to have an epilepsy show normal EEG. patterns. Far from being a 'missed"

diagnosis, the presence of a normal EEG. with a known lesion is of great clinical significance.

For the space occupying lesions the normal EEG. suggests either:

1) that the tumour is invading a very small area of cerebral cortex,
2) that the tumour is invading white matter only.
3) that the tumour is invading a remote subcortical nuclear structure,
4) that the tumour is not disturbing cortical or subcortical function.

A normal EEG. in a patient with an epilepsy suggests either:

1) that some extracranial cause has been activating the brain to produce the ictal or convulsive state,
2) that the lesion causing the epilepsy is deep to the convexities of the hemispheres, being hidden in a sunken gyrus or the under surface of the cerebrum,
3) that the lesion is in a subcortical nuclear system not projecting to the cortex.

Activation techniques, including sleep[2], increase the incidence of abnormal records in epileptics to around 90% and assist in differentiating the above possibilities.

It has been shown by Abbott and Schwab[3] that epileptics with normal EEGs. tend to respond to anticonvulsant therapy more satisfactorily and to have a better ultimate prognosis.

Many conditions affecting the nervous system show a high incidence of normal EEG. patterns. There is little point in enumerating the various conditions for the normal EEG. suggests:

1) that there is no lesion affecting the cerebral hemispheres,
2) that a lesion, if present, is of very small size,
3) that a lesion is remote from the recording electrodes,
4) that a lesion is only affecting white matter,
5) that a lesion has not caused cortical or subcortical dysfunction and requires activation by some exogenous conditions before abnormal patterns appear.

* * *

The Abnormal Electroencephalogram

The abnormal EEG. is evidence of abnormally functioning grey matter. The lesion producing the abnormal activity may not be demonstrable by any other clinical techniques or be evident at autopsy.

The significance of the abnormal EEG. in the 5 to 7% of normals is not fully understood. However, it has been shown that these individuals break down psychiatrically or convulse under lesser conditions of stress that those with normal EEGs. The "falsely" abnormal pattern is not specific to electroencephalography it is found in virtually all clinical tests, for example some 4% of a group of clinically normal persons undergoing routine medical examination have shown an abnormal electrocardiogram[4] and it is even reputed that some 3% of normals show an extension of the big toe on plantar stimulation[5].

The Causes, Locations and Types of Abnormal Patterns

The abnormal EEG. may be produced by extracranial or intracranial processes. Among the extracranial processes are hypoglycemia, hepatitis[6], pernicious anemia[7], severe burns[8] and Addison's disease[9]. Presumably these conditions alter some component of the blood in such a way that cerebral metabolism becomes interfered with. The abnormal patterns produced by extracranial causes are rarely of a specific nature, but are of some value in differential diagnosis and may be used in following the course of the disease.

The intracranial causes of the abnormal EEG. may be cerebro-vascular, space occupying, degenerative, toxic or atrophic lesions. The abnormality in the EEG. is produced by the cortical or subcortical grey matter and the abnormal pattern may be the result of:

1) depression of normal metabolism of the grey matter,
2) irritation of the grey matter,
3) suppression of cellular function by pressure, trauma or destruction.

The abnormal activity may be seen:

A) Locally in one hemisphere (localized, unilateral),
B) Locally in both hemispheres (localized, bilateral, symmetrical or asymmetrical),
C) Widespread over one hemisphere (widespread, unilateral),
D) Widespread over both hemispheres (widespread, bilateral, symmetrical or asymmetrical)

and the pattern may be made up of one or more of the following patterns (figure 2):

Figure 2 Typical EEG Patterns

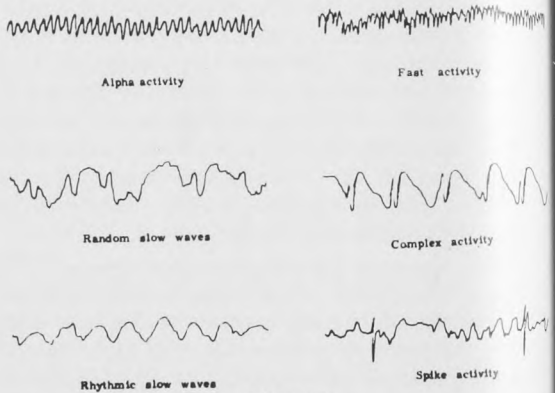

Figure 2
The alpha activity is normal. The other patterns are abnormal in the adult.

i) Random Slow Waves,
ii) Rhythmic Slow Waves,
iii) Fast Activity,
iv) Complex Activity,
v) Spike Activity.

From the type of pattern and its distribution it is possible to deduce the form and origin of the dysfunction of the grey matter. It is not necessarily possible to deduce the etiology as many dissimilar conditions may disturb cellular function in the same way.

The Significance of the Different Abnormal Patterns

A) **Localized, Unilateral Abnormalities.** An EEG. abnormality localized on one hemisphere is produced by a pathological process in that hemisphere. The finding of localized abnormality should not be ignored without further clinical investigation.

i. Random Slow Waves localized to one area are produced by a process depressing metabolism of the cortical neurons in that area. The pathological cause may be a tumour, cerebral abscess, encephalitic patch, intracerebral hematoma, vascular occlusion or cortical atrophy. Almost invariably air encephalography, ventriculography or angiography will demonstrate the lesion producing the random slow waves. The slow waves do not come from the lesion but the cortical cells around the lesion. The EEG. will indicate the functional extent of the lesion and add information not necessarily obtainable clinically and radiologically.

ii) Rhythmic Slow Waves arise from cortical or subcortical areas. Localized unilateral patterns at the cortex arise from long standing or slowly progressive lesions. Examples are scar formations and some tumours. Rhythmic patterns projected to one area of the head arise from lesions in the basal, thalamic or reticular nuclear systems. It is often possible to differentiate cortical from subcortical lesions by the effect of arousal stimuli on the pattern[10]. Cortical patterns are unchanged by the stimuli and subcortical patterns usually modified. There may be no radiological evidence of the lesion producing the slow waves. This may be due to the lesion being a small atrophic process. The absence of radiological evidence does not exclude the presence of a lesion. Local rhythmic slow waves may be associated with one of the epilepsies.

iii-v) Fast, Complex and Spike Activity seen locally in one hemisphere may be cortical or subcortical in origin. The causative lesion may not be demonstrable radiologically. The characteristics are similar to those of localized rhythmic slow waves but almost invariably these patterns are associated with an epilepsy. The epilepsy may not show up in the classic convulsive form and may not be evident except by careful questioning. Paroxysmal autonomic or emotional disturbances may occur. Temporal lobe abnormalities are frequently associated with episodic psychiatric disorders.

B) **Widespread, Unilateral Abnormalities.** Widespread abnormality involving one hemisphere is virtually pathognomonic of organic brain disease affecting that hemisphere. The significances are similar to those of localized abnormalities but usually indicate more severe clinical states.

i) Random Slow Waves widespread over one hemisphere indicate widespread cortical involvement. The causative lesion will usually be demonstrable clinically and radiologically. Occasionally there are slow waves over one hemisphere and a suppression of activity over the other hemisphere, in this case the side of the suppression shows the side of the lesion.

ii) Rhythmic Slow Waves all over one hemisphere arise in the unilaterally projecting subcortical nuclei. The anatomy of these nuclei is inadequately known. More frequently both rhythmic and random activity appear together in the EEG. This suggests that the lesion is involving both the cortex and the thalamic, reticular or basal nuclear systems. The prognosis is usually unfavourable in these cases.

iii-v) Fast, Complex or Spike Activity seen widespread over one hemisphere is a rare finding. The pattern may arise cortically or subcortically. The site may be deduced from the characteristics of the pattern and the effects of arousal stimuli. The causative lesion may be an atrophic process or tumour.

C) **Localized, Bilateral Abnormalities.** Bilateral abnormalities may be symmetrical or asymmetrical in pattern and amplitude. When symmetrical the lesion is usually in those subcortical nuclei projecting to both hemispheres. When asymmetrical the side of the greater abnormality is usually the side of the lesion.

i) Random Slow Waves appearing bilaterally in similar localized areas are too rare a finding to merit comment.

ii) Rhythmic Slow Waves appearing locally, bilaterally and symmetrically are the most common EEG. abnormality. The rhythmic slow waves project to similar areas of both hemispheres from the mid-line reticular and thalamic nuclei. These nuclei are made up from multi-neurone chains which are very susceptible to metabolic interference[11]. Vasoconstriction from overbreathing, anaesthetic agents, edema from a tumour or vascular accident, hypoglycemia and some exogenous metabolic abnormalities will produce bilateral rhythmic slow patterns. An atrophic lesion will produce bilateral patterns and is usually associated with an idiopathic or cryptogenic epilepsy. So many different agents will cause the bilateral

rhythmic activity that it may be regarded as a non-specific response of the brain. Rhythmic slow activity suggests the presence of an organic basis for the clinical state. Usually it cannot suggest the etiology.

iii-v) Fast, Complex or Spike Activity seen bilaterally appears to arise from the midline nuclear systems. They are produced by electrically active lesions and are almost invariably associated with an epilepsy.

D) **Widespread, Bilateral Abnormalities.** These patterns are frequently difficult to analyze. They may be symmetrical or asymmetrical. The symmetrical patterns suggest involvement of the bilaterally projecting subcortical nuclei. When asymmetrical the side of the greater abnormality is usually the side of the lesion.

i) Random Slow Waves may be equal in amount but not in appearance over both hemispheres. It may be difficult to detect the side of the great abnormality. Usually bilateral random activity is produced by bilateral cortical disease. This may be an encephalitis, bilateral subdural hematomata, a tumour spreading from one hemisphere to the other, cortical degenerative process and some other diseases. The presence of the bilateral widespread random slow activity is usually of unfavourable prognostic significance. Random and rhythmic slow patterns appearing together indicate cortical and subcortical involvement.

ii) Rhythmic Slow Activity all over both hemispheres is usually symmetrical. It is a non-specific response of the brain to many different conditions. When widespread the causative condition is involving more resistant nuclear systems than with the localized bilateral patterns.

iii) Fast Activity of low voltage from all areas of the head is found in some normals. After administration of barbiturates higher voltage fast waves dominate the EEG. as the drug takes effect and as its effect wears off. This barbiturate activity may be evident two days after administration of the drugs. As high voltage fast activity is seen in some epileptics care must be taken to withhold the barbiturate drugs before the EEG. test or to provide data concerning their administration.

iv-v) Complex and Spike Activity which appears from all regions of both hemispheres symmetrical is virtually always associated with some form of epilepsy. In some cortical degenerative processes widespread low voltage asymmetrical spike patterns appear.

Summary

The electroencephalograph is not an instrument that writes a diagnosis on a sheet of paper. It is an instrument which indirectly indicates the way disease processes are affecting the functioning of the nuclear matter of the brain. Correlation between the suggestive electroencephalograph findings and the clinical state is essential before the test can be used to its full value.

The electroencephalogram has certain definite limitations. It is unable to detect abnormality in some portions of the grey matter of the brain and of lesions in the white matter. The knowledge of the limitations can be turned to value by the clinician when he wishes to arrive at an anatomical diagnosis or to determine whether the brain is functioning normally in the presence of a known lesion.

When used purely as a diagnostic guide the EEG. can only state the presence or absence of different conditions in terms of probability. In this respect the test differs in no way from any other clinical procedures.

The significance of the EEG. findings are best worked out by discussion between the clinician and the electroencephalographer and not by relying purely on the written report. Using the report as a basis for discussion makes the EEG. an extremely valuable test for the clinician who requires to assess the degree of abnormal cellular function produced by the disease process.

Acknowledgments

I wish to acknowledge with thanks permission from the Medical Superintendent, Hospital for Mental Diseases, Brandon, to print figure 1. Figure 2 was taken from records at the Winnipeg General Hospital.

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Pathology

Blood Alcohol Determinations

J. C. Wilt, M.D., E. Hirst, B.Sc., and D. Nicholson, M.D.*

The increased awareness of the legal profession and the general public of the hazards associated with "drinking while driving" makes desirable a more accurate assessment of actual alcoholic consumption than is gained by the general impression of a constable or bystanders.[1]

The purpose of this paper is to outline a technique which, with certain modifications, has proved satisfactory in the analysis of 400 blood samples in the course of the last 7 years.[2] Most of these blood samples were collected and submitted together with history and physical examination by the personnel of the Psychopathic Hospital in Winnipeg.

Collection of the Blood Sample

The legal implications associated with the collection of blood for this particular test require that certain precautions be taken in order that the results of the test be not invalidated.

1. Before the physician collects the blood sample the patient should be informed about the nature of the test and his written consent obtained for the removal of the blood. Should medical examination indicate necessity for a blood alcohol analysis, i.e.—differentiation of a head injury from a state of alcoholic intoxication—this precaution is unnecessary.

2. **No** alcohol including tinctures, etc., may be used during any stage of the collection of the blood sample. The arm is cleansed with an aqueous germicide and following removal of the needle from the vein a dry sterile swab is placed over the site. No alcohol is to be used either for the preliminary "sterilization" or for the rinsing of syringe and needle.

3. The most satisfactory method of obtaining a 5 ml. blood sample for alcohol analysis is by means of a special Keidel blood letting tube containing an anti-coagulant. A fairly satisfactory alternative is to collect 5 ml. of venous blood with a syringe, the blood to be expressed immediately upon removal into a thoroughly cleansed and sterilized bottle containing an anti-coagulant (potassium oxalate).

4. The blood specimen should be labelled immediately, as follows:

a) Name of the patient.

b) Date and hour of collection of blood sample.

c) Signature and address of the physician.

5. The method used to attach the label to the specimen container is of prime importance. A gummed label is required and should be affixed below and then over the cap of the container in such a manner that a seal is formed which must be broken if the container is opened.

6. The requisition submitted with the blood sample should also be accurately filled in and then securely attached to the specimen. Entered on the requisition should be the name and address of the person to whom the report is to be sent; in addition any pertinent clinical information should be entered, in particular stressing any possible medico-legal action.

7. When medico-legal action is implied, continuity in transmission of blood for alcohol determination must be established. Several methods for transmission have proved satisfactory:

a) The analyst himself withdraws the blood sample and is also responsible for its transmission. Unfortunately this arrangement is seldom possible.

b) The physician withdrawing the blood sample himself delivers it to the analyst. It must be stressed that under such circumstances the physician is then responsible for any tampering with the specimen while it is in his possession. It is therefore recommended that the specimen be kept upon the person of the physician, or if that is not feasible the sample should be placed in a locked compartment. The alcohol in a sample of blood will show no depreciation for at least 48 hours following collection if it is kept at room temperature in a sealed container.

c) The physician collects and labels the blood sample under the direct supervision of the police. The police then transmit the sample directly to the analyst.

d) Mailing—when a blood sample is mailed to the analyst the doctor collecting the blood must personally place sample in a mailing container, take it to the Post Office, and dispatch it to the analyst by registered mail. The analyst must personally open the wrapper and keep it for identification in court.

The technique to be described in detail involves the same principles as the method outlined by Cavett.[4]

Principle

The mixture of potassium dichromate and sulphuric acid produces chromic acid which in turn oxidizes any alcohol diffused from the blood. The remaining unoxidized chromic acid is determined by titration with a reducing agent and indicator and compared with a known negative control test.

Glassware

All glass ware used for blood alcohol analysis must be chemically clean and must be kept for that purpose alone.

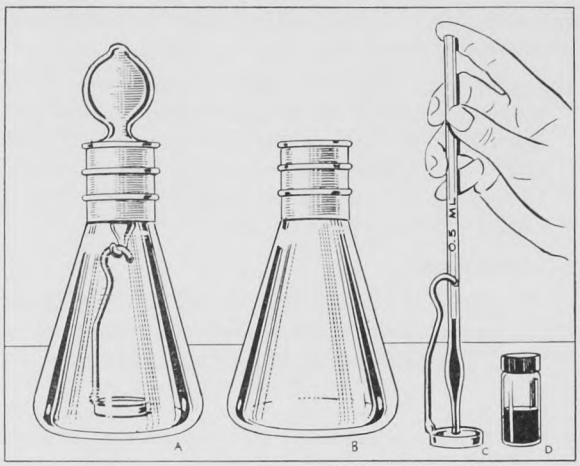
*From the Department of Pathology, Winnipeg General Hospital.

Required

1. Two 500 ml. Erlenmeyer flasks with ground glass orifice.
   (Catalogue No. 6900, Ace Glass Inc., Vineland, N.J.)
2. Ground glass stoppers with hooks, to fit Erlenmeyer flasks.
   (Catalogue No. 8265, Ace Glass Inc., Vineland, N.J.)
3. Glass cups with hooks to hang from Erlenmeyer flask stoppers.
   (Ace Glass Inc., Vineland, N.J.)
   Filter paper suspended from a rubber stopper has been recommended, but is not satisfactory.
4. One 5 ml. reversible burette. (Not necessary but exceedingly convenient).

Reagents

1. Concentrated sulfuric acid—Merck's—Reagent grade.
2. Standard dichromate solution—Merck's—Reagent grade.
   Potassium dichromate ..... 0.852 grams.
   Dissolve in distilled water.
   Dilute with distilled water to a final volume of 1 litre.
3. **Methyl Orange Solution**
   a) Methyl orange ..... 0.1 gram
   b) N/10 NaOH ..... 25.0 ml.
   Place in 100 ml. volumetric flask and shake to dissolve.
   Add distilled water to a final volume of 100 ml.
4. **Ferrous Sulfate Solution**
   a) Ferrous sulfate (Merck's-Reagent grade) ..... 5 grams
   b) Distilled water ..... 15 ml.
   Dissolve and add:
   c) Concentrated sulfuric acid ..... 3 ml.
   d) Add distilled water to a final volume of ..... 25 ml.
   (The above solution when freshly prepared is a crystal clear green color: in 6 to 8 weeks a brownish tint develops which yields a poor end-point in the titration).
5. **Reducing Fluid**
   Place a 50 ml. volumetric flask in cold running water.
   Add reagents in the following order—
   a) Distilled water ..... 16 ml.
   b) Concentrated sulfuric acid ..... 16 ml.
   c) Methyl orange solution ..... 15 ml.
   d) Ferrous sulfate solution ..... 1 ml.
   e) Distilled water to a final volume of ..... 50 ml.
   (As a considerable degree of heat will be generated when all ingredients are combined, the flask should be held in cold running water; agitation should be gentle at first, then mix well).
   f) Cool solution to room temperature and make up to a final volume of 50 ml. Mix thoroughly. This solution will keep for 7 days.



. **Positive Controls**
   Absolute ethyl alcohol ..... 1 ml.
   (Specific Gravity 0.8)
   Distilled water to ..... 100 ml.
   Add exactly 0.5 ml. of the above 1% solution of alcohol to 3.5 ml. of blood—give 0.100 gm. %.
   Add 1 ml. of the 1% alcohol to 3 ml. of blood to give 0.200 mg. %.

Procedure

1. Pipette 10 ml. of standard dichromate solution into each of the two Erlenmeyer flasks.
2. Pipette 10 ml. of concentrated sulfuric acid into each of the two Erlenmeyer flasks.
   Mix well.
3. Pipette slowly into one glass cup 0.5 ml. of blood sample.*

*We recommend an ordinary Mohr type 1 ml. pipette with narrow tip. Empty from 0 graduation to 0.5 ml. over a period of 120 seconds, not less, at a uniform rate to allow the viscid blood to flow off the walls. Only 2.5 cu. mm., mostly plasma remains. This is within a cubic millimeter or two of the water adhering after a 15 second delivery as used in official standardization. If the pipette is emptied of blood in 15 seconds, 7.5 cu. mm. adheres, lowering the result by one per cent. This will be further increased with plethoric bloods having high red cell counts and plasma protein which augment their viscosity. Tiny clots, cotton fibres, air bubbles and other foreign particles interfere with delivery. Using any technique with lipemic blood, fat droplets float to the top, adhere to the wall surrounded by a pink halo of red cells. Letting 0.5 ml. of blood pour out of an ordinary Ostwald or serological pipette in one to two seconds may give a result 8 per cent too low.

The advantages of the Ostwald type pipette are more assumed than actual and they are nullified by substantial shortcomings.

Conway, E. J., Microdiffusion Analysis and Volumetric Error. 3rd Edit., 1950, Chapter V on Pipettes.

The reduced area of the internal surface is offset by poor drainage from the sloping walls of the bulb, many of which approach the globular. With the opaque meniscus of blood, the top of the red column is usually placed behind the zero line and this reduces the delivery in the 0.5 ml. Ostwald type pipette by 1½ per cent.

A normax 1 ml. Mohr type pipette with its fine delivery jet made by Kimble and certified by the U.S. Bureau of Standards performs well but the cost at $5.00 to $6.00 is too high for routine use. Practically all pipettes on the market for routine use today are carefully calibrated but they have stubby tips and open jets. Calibrations are carried out using distilled water and they deliver less blood unless special precautions are taken. The result depends more on the pipettor than on the pipette.

4. Suspend this cup from the hook on stopper and stopper flask.
   The other flask is stoppered and constitutes the blank.

5. Label each flask with patient's name and laboratory number.

6. Leave both flasks at room temperature for a minimum period of 10 hours, then titrate with the reducing fluid to the first permanent pink tint. An alternative which is not so satisfactory is to heat both flasks at 70°C. for 2 hours, cool and then titrate.

7. Calculation:

S=amount of titrating fluid used for the control blank flask.
U=amount of titrating fluid used for blood alcohol analysis flask.

$$\frac{S - U \times 0.4}{S} = \text{grams of alcohol per 100 ml. of blood}$$

or

$$\frac{S - U \times 400}{S} = \text{milligrams of alcohol per 100 ml. of blood.}$$

If, following the calculation, 250 mgm. or more of alcohol are found per 100 ml. of blood the test should be repeated, using 0.25 ml. of blood in place of 0.5 ml.

The following formula is then used:

$$\frac{S - U \times 400 \times 2}{S} = \text{mgm. of alcohol per 100 ml. of blood.}$$

Table I shows the results obtained on sample groups of 6 normal people, 6 diabetic patients and on 8 specimens of blood to which had been added a measured amount of alcohol.

Table I

Results on 6 normal people gm/100 ml.	Results on 6 diabetic patients with elevated blood sugar gm/100 ml.	Results on known samples Measured amounts of Alcohol added to normal blood gm/100 ml. Known Concentration	Analysis 1	Analysis 2
0.010	0.028	0.1	0.095	0.093
0.013	0.0	0.1	0.092	0.094
0.004	0.0	0.2	0.19	0.188
0.003	0.012	0.2	0.19	0.192
0.002	0.015	0.3	0.28	0.282
0.005	0.010	0.3	0.285	0.280
		0.4	0.378	0.380
		0.4	0.382	0.385

Other methods giving similar results — gm. alcohol per 100 ml. blood:

This technique by J. C. Wilt	Conway procedure — Thiosulphate titration by R. Hunter
0.136	0.138, 0.134, 0.138
0.211	0.216, 0.203, 0.212, 0.212
0.009	0.007

The large surface of the ground stopper gives a more substantial seal than the top margin of the standard Conway unit. The more recent smaller unit 2B has a flanged top to give a larger sealed area.

Interpretation of Test

This particular test identifies alcohol in the blood as an oxidizable substance only; non-alcoholic oxidizable substances do occur at times in the blood in health, usually 0.005 or 0.010 gm. per 100 ml. of blood is demonstrated, although occasionally a level of 0.020—0.025 gm. of oxidizable material per 100 ml. of blood may be found.

Certain diseases such as diabetes are accompanied by presence of acetone in the blood which is an oxidizable reducing substance. In our experience this does not give rise to a level above 30-35 mgms. per 100 ml., even in the presence of severe diabetes.

The National Safety Council of the American Medical Association considers 0.150 gm. of alcohol per 100 ml. of blood as conclusive evidence of drunkenness; this figure has been accepted in several countries. At a level of 0.350 to 0.450 gm. of alcohol per 100 ml. of blood, stupor or coma develop which may terminate in death; at this stage the patient is particularly susceptible to aspiration of regurgitated stomach contents with possible asphyxiation.

The analysis of blood for the presence of alcohol only indicates the amount of alcohol in the sample at the time of collection. However when an accident has occurred several hours prior to removal of the blood a knowledge of the blood alcohol level at the actual time of the accident is necessary. In the body, alcohol is oxidized at a fairly constant rate of 0.010-0.015 gm. per 100 ml. of blood per hour, therefore (if no further alcohol was consumed in the interval) an approximation of the minimal alcohol level in the blood at the time of the accident may be reached by multiplying 0.010 gm. by the number of hours in the interval from the time the accident occurred to the time the blood sample was withdrawn, then adding this sum to that already obtained in the analysis.

As a rule the alcohol content of urine cannot be correlated as closely with the degree of intoxication as the blood alcohol level, but the test when used as an adjunct of the blood alcohol analysis, is occasionally of value to determine the duration of the drinking and whether the blood alcohol level at the time of the analysis is decreasing or increasing. The principal advantage gained by submitting a specimen of the patient's urine with the blood sample is that a sugar and acetone analysis can be readily carried out in conjunction with blood alcohol analysis.

Conclusions

A satisfactory technique for blood and urine alcohol analysis has been outlined. The methods of collection and transmission of the blood sample and the interpretation of the result has been discussed.

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Orthopedics

Some Common Foot Conditions of General Concern

A. P. Guttman, M.D., F.R.C.S. (Edin.) F.I.C.S.

At all ages foot disabilities constitute a fair part of medical practice. Various pathological entities interfere with the proper function of the feet which normally provide a stable support for the body weight in standing, and a resilient spring by which the body is propelled forward on walking.

Some of the common foot conditions as met with in daily practice together with their treatment is briefly described:

Bunion

A bunion or hallux valgus consists of an extreme adduction of the proximal phalanx of the great toe towards the midline of the foot and includes the adventitious bursa over the metatarsal head plus a corn or callosity in the skin. In its extreme form it is associated with varying degrees of varus of the first metatarsal bone. Lesser degrees of deviation of the first phalonx are not uncommonly due to badly designed and illfitting shoes in the presence of a normal first metatarsal bone. Once a bunion develops the original deviation is progressively increased by the contracture and shortening of the tendons so that eventually the base of the first phalanx is displaced laterally articulating with the lateral condyle of the metatarsal head, leaving the medial condyle as a prominence on the medial side of the foot which becomes subjected to pressure and friction from the shoe. A bursa forms over the metatarsal head and a corn in the skin. The cartilage on the medial side of the head of the first metatarsal degenerates and marginal bony osteophytes form, resulting in osteo-arthritis of the metatarso-phalangeal joint.

Treatment may be:

(a) Conservative
(b) Operative

(a) Conservative Treatment: Mild cases may be treated by properly fitting shoes with a straight inner side. A metatarsal bar may be added to relieve weight bearing trauma. In old people adhesive strips may be placed around the bunion to relieve the pressure of the shoe on the bunion. An inflamed bursa overlying the bunion (bony part) requires the usual treatment of an acute inflammation with rest in bed.

(b) Operative Treatment: This is indicated for the relief of symptoms and in women for correction of deformity. It should not be done in the presence of acute inflammation. The operation recommended as the most satisfactory is the Keller in which the proximal half of the first phalanx is removed together with the exostoses about the metarsal head. A plaster cast covering the forefoot and big toe with the metarsal arch moulded and the big toe in normal anatomical position is worn for two weeks. The sutures are then removed and another similar cast applied for three to four weeks during which time weight bearing is permitted.

Hammer Toe

A hammer toe usually affects the second toe and consists of a dorsiflexion of the proximal phalanx, plantar flexion of the second and either flexion or extension of the distal phalanx. The toe cap of the shoe presses on the head of the first phalanx resulting in a painful corn, and a bursa forms underneath this over the interphalangeal joint. This latter often becomes inflamed and may even form an abscess. A corn may form at the tip of the toe from pressure against the sole of the shoe. The condition is usually bilateral and most commonly due to crowding of the toes into poor fitting shoes. It may be congenital. It is commonly associated with hallux valgus and is due to the displacement of the latter forcing the second toe into a flexed position.

Treatment may be:

(a) Conservative
(b) Operative

In young children the condition can be corrected by manipulation and the application of one-half inch adhesive strips around the adjacent toes and over the dorsum of the second or other affected toes so that the hammer toe is in line with the others. In older children and adults the deformity becomes fixed due to contraction of the ligaments and operation is required. The corn is excised, the contracted ligaments are divided and the first interphalangeal joint fused.

Morton's Metatarsalgia

This condition is characterized by pain in the sole of the foot beyond the metatarsal necks when the foot is in use. The pain disappears when the foot is at rest. Pain on firm upward and backward pressure in the sole just distal to the third and fourth metatarso-phalangeal joints is the most constant physical sign. Numbness in the adjacent toes is usually present. Pathologically there is a fibrosis surrounding the involved digital nerve associated with endarteritis of the interdigital arteries.

In early cases wearing of metatarsal bars may give symptomatic relief but in severe and long standing cases resection of the thickened plantar digital nerve gives complete relief in almost all cases.

Plantar Fasciitis

Pain on the inferior surface of the heel on standing with tenderness on pressure may be

caused by a variety of conditions. When there is an obvious cause present, it is easy to treat. Where X-rays are negative, it may be due to fibrositis, or foot strain. In some cases neurosis may be a factor. These cases are often difficult to clear up. Local injections of procaine to the trigger point repeated as required clear up the pain in many cases. Wearing of soft rubber insoles and heels relieves the pain. In some a raise on the heel of the shoe relieves the pressure of the heel on weight bearing. Hydrocortone by injection to the affected site may be employed. In the event that conservative treatment fails the origin of the plantar fascia and short muscles are separated from the under surface of the calcaneus.

Flatfoot

The structures which preserve the long arch of the foot are the shape of the bony components, the plantar ligaments and the postural activity of the tibial group of muscles.

Flat foot may be:

1. Congenital
2. Acquired

Congenital type may result from anomalies of the tarsal bones or failure of the tibial muscles to acquire the necessary postural tone to preserve the arch.

Acquired type may be:

1. Osseous
2. Ligamentoses
3. Muscular

Osseous variety may result from injury or disease.

Ligamentous variety may follow rupture or avulsion of the plantar ligaments from their attachments.

Muscular type may be:

1. Paralytic
2. Spastic
3. Postural or static

The static type is the most common and arises when the postural muscles of the longitudinal arch are unable to fulfil their function, such as occurs following:

1. Convalescence after illness or childbirth due to loss of muscle tone.

2. Excessive fatigue of normal muscles as in certain occupations requiring prolonged standing.

3. A rapid increase in body weight such as often occurs at the menopause.

Examination of the foot—an inspection of the shoe will show excessive wear of the sole and heel on the inner side with bulging of the inner side of the upper.

On standing bare foot the upper part of the calcaneus points medially while the lower portion points laterally. There is loss of height of the longitudinal arch with a bulge on the medial aspect of the foot due to the prominence of the head of the talus or navicular.

Tenderness is often present on both the medial and lateral aspects of the ankle.

Treatment: Some cases of flat foot have no symptoms.

In children with a mobile foot the inner aspect of the heel and the inner or outer aspect of the sole of the shoe is raised and an arch support placed under the insole to raise the longitudinal arch. Foot exercises to strengthen the tibial and short foot muscles are prescribed. These cases in children should be watched over a period of years.

In adults with symptoms of foot strain an arch support of leather and rubber is made for the individual patient and is worn inside the shoe.

Plantar Wart

This occurs on the sole of the foot and is caused by a filtrable virus. It is infectious. It is common, quite annoying and resistant to treatment because of its location. It is usually single and is most commonly confused with corns and calluses. The plantar wart is in the skin and may be surrounded by a zone of callus. Treatment for this condition is multiple.

The common methods are:

1. X-ray
2. Surgical excision
3. Electrocoagulation or electrodesiccation under novocaine anaesthesia, the crust being removed in ten to fourteen days.

Abstracts

Complications Following the Use of Efocaine: D. C. Moore, Surgery, 35: January, 1954, 109.

In this article the author discusses various complications that have followed the use of efocaine for producing prolonged relief of pain. Dr. Moore reports not only his own complications but also submits the findings of many others who have experienced untoward reactions following the use of efocaine.

Efocaine is a mixture of 1% Procaine base and 0.25% Procaine hydrochloride as the anaesthetic base, 5% butyl-p-amino benzoate (butesin), 2% polyethylene glycol-300 and 78% propylene glycol as the aqueous-miscible solvent, and 0.1% sodium metabisulfate and 1:2500 phenyl mercuric borate as the preservative.

Maykut[1] has shown "that the prolonged action of the long-lasting local anaesthetic substances, presently on the market, is not due solely to the anaesthetic substances present—but appears to be associated with the tissue destruction produced by other ingredients present in the commercial preparations."

Damage to the spinal cord may occur even if the nerve is injected several inches away from the root and under direct vision. The perineural

spaces of any peripheral nerve, while essentially filled with loose areolar tissue, etc., are still continuous with those of their roots at least as far as the spinal cord, and the solvents in the efocaine may track along this pathway to the spinal cord, and produce necrosis with resulting myelitis.

1. Maykut, Madelaine O.: Proceedings of The Canadian Anaesthetists' Society, 1953, 5.

Max Minuck, M.D.

A Technique for Tracheo-Bronchial Toilet in the Conscious Patient: David Zuck, M.B., Ch.B., D.A. Anaesthesia 6: 226, October, 1951.

A nerve block of the superior laryngeal nerve is done as follows: 50 mgm. Demerol is given 1 hour before the procedure. An abdominal binder is applied and the patient is placed in a sitting position with a pillow behind the neck and the head extended. He is told not to talk, cough or swallow. A wheal is made over the thyroid notch with 2% novocaine. Holding the hyoid between thumb and index finger of the left hand, insert an 8 cm. needle through the wheal and inject as it is advanced along a line towards the cornu of the hyoid until it reaches a point slightly below and anterior to the cornu. Inject 2-3 c.c. 2% novocaine with the needle moving. Inject another 2 c.c. as the needle is withdrawn. Using the same wheal, repeat on the other side. If the needle goes beyond the cornu it may penetrate the great vessels of the neck. Spray the clearer nostril with 5% cocaine, using less than ½ c.c. A No. 7 Magill endotracheal tube is then lubricated and passed into the nasopharynx. Tell the patient to close the lips and breathe through the nose, and hold the other nostril closed. Manipulate the tube and head till the tip of the tube is at the opening of the glottis. Attempt intubation of the trachea with a piece of gauze over the end of the tube. Entry causes coughing, but tolerance to the tube develops rapidly. Suction repeatedly till the chest is clear, allowing the patient to rest between insertions of the suction catheter. If bronchospasm is present, begin a slow intravenous drip of aminophylline, giving up to .25 g. No food or drink must be taken for two hours.

This technique described by Dr. Zuck has been used increasingly at St. Boniface Hospital, and it is one of the most useful things I have come across in years. It is not a difficult procedure. Once the tube is in place the patient can relax comfortably. It is then just a matter of time before the chest is completely cleared of secretions. Posturizing may help in cases of atelectasis, and a gum elastic catheter may be bent near the end so as to facilitate its entrance into right or left bronchus. Coughing may be encouraged and it is not harmful since excessive pressure cannot be produced with the glottis held open by the tube.

I would like to recommend this procedure and urge you to try it for the wet chest or atelectasis.

M. R. Bennett, M.D.

Preoperative Anaesthetic Out-Patient Clinic: Loder, R. E., and Richardson, H. J.: Lancet, 166: June 5th, 1954, 1177.

A pre-operative consultation with the anaesthetist (exclusive of the immediate pre-operative visit) was first suggested by Lee in 1949, and since then several hospitals in England have been holding pre-operative clinics with decided success.

The authors analyze 500 cases seen at such a clinic held at the Peterborough Memorial Hospital at Northants. The patients seen were all the surgical waiting list and aged more than 40, all those who were to undergo major surgery irrespective of their age, and all those referred to the clinic by the surgeons before they were placed on a waiting list. A history was taken which included previous operations and anaesthetics, and previous blood transfusions. Included in the physical examination were estimation of the vital capacity, breath holding tests and chest expansion. The patient was given advice on smoking, dieting, etc. After this the patient's risk was assessed (this might only be preliminary). If necessary the patient might be referred to other departments, cardiologist, physiotherapist, etc.

Of the 500 cases—226 males and 274 females—318 patients were considered fit for anaesthesia for the proposed operation, 22 were considered unfit for any form of anaesthesia for the proposed operation, and 142 were considered fit for some suitable form of anaesthesia following investigation and treatment.

Of those considered unfit 15 were in heart failure, 2 with severely impaired coronary circulations, 1 in uremia, and 4 with severe bronchitis, bronchiectasis and emphysema.

The advantages of such a preoperative examination by the anaesthetist are numerous.

To the Patient—He is reassured. He is further saved a good deal of expense by having the investigation and some of the treatment done outside of the hospital.

To the Surgeon—The surgeon has the convenience of knowing that a patient coming to the hospital for major surgerey, is considered fit to have the operation done at once.

To the Anaesthetist—It is the anaesthetist's duty to examine personally each patient to whom he is going to give an anaesthetic. It is easier to discuss a case with the surgeon while the case is on the waiting list than to suggest treatment or further investigation for a patient that is slated for the following day.

Max Minuck, M.D.

Clinico-Pathological Conference

Deer Lodge Hospital

February 3, 1953

Miss I. J. White, Canadian, date of birth Oct. 17th, 1880. Occupation, Nursing Sister.

This patient had served in World War I during which time she developed a tuberculous keratitis for which she was given a pension.

She remained in fairly good health until the latter part of 1943 when she started to develop dyspnoea and oedema of ankles and was admitted for treatment to Deer Lodge Hospital in April, 1944. She was diagnosed as Arteriosclerotic Heart disease with mild hypertension. She improved with treatment and was discharged after a week's hospitalization.

On May 3, 1947, she was re-admitted to this hospital for further treatment of her eyes and also for a general feeble condition. On admission patient was confused and irrational. She had bladder incontinence and required enemas every three days.

May 10, 1947—**Consultant's Report:**

She is confused and dis-orientated, there is a fine lateral nystagmus; fundi were not seen, pupils irregular with dense opacities; there is a suggestion of bilateral facial paresis; ankle and knee reflexes are present, equal; absent abdominal reflexes, there is however a bilateral plantar extension.

The lungs are resonant, breath sounds are distant, there are no rales. Heart, 11 cms. diam., sounds are loud; late systolic murmur. BP 130/??.

Liver is markedly enlarged, slightly tender. Spleen not palpable, moderate oedema of ankles.

Impression: Patient has definite cardiac disease of the hypertensive and arteriosclerotic type, also evidence of a diffuse brain lesion, presumably vascular in origin. However, the picture was not quite clear.

May 13, 1947—E.C.G.—Interpretation—Chronic Left Ventricular Strain.

May 19, 1947—Internist report read as follows: "She is totally disorientated, but has glimpses of intelligence, she has no asterognosis. Her speech is clear but her ideation is not. At times she shows evidence of oproxia. There is bilateral nystagmus, a suggestion of bilateral facial weakness and 12N weakness. Marked motor weakness and bilateral extensor plantar. This must be a diffuse cerebral degeneration with evidence of both cortical and bulbar lesions, the oedema is at least in part associated with hypoproteinemia and one wonders if thiamin deficiency is related.

July 15, 1957—Patient responds to questioning but is confused and retarded. B.U.N. 10 gm.%; Plasma Proteins: T.P. 5.87 gm.%; Alb. 282 gm.%; Glob. 3.05 mg.%; N.P.N. 23 mg.%. No spinal puncture ever done. Sed. Rate 52 mm; WBC 7,300; Hgb. 80%; RBC 4,380,000.

October 3, 1947—This elderly female with advanced cerebral arteriosclerosis, hypertension and coronary disease continues downhill course; she does not move, is difficult to arouse, and requires considerable nursing care.

December 10, 1947 — The patient's condition deteriorating and is difficult to rouse; she sweats profusely.

December 23, 1947—X-ray of chest—considerable osteoporosis, otherwise negative.

Patient died January 3, 1948.

Pertinent Autopsy Findings—A 351

Cranial Cavity

The skull is dense and thicker than usual. The brain surface is hyperaemic. There appears to be A cyst in the right cerebrum and there is a cyst above the stalk of the hypophysis.

On sectioning the the brain a tumor mass is found in the centre of cerebellum extending into lower part of pons and upper end of medulla. The mass has a reddish coloration.

The lower surface of cerebellum shows conus formation over foramen magnum.

The lateral ventricles are very greatly distended to an antero-posterior measurement of 11 cms. and are filled with clear C.S.F.

Thoracic Cavity

The heart weighs 340 gms. The mitral valve Shows hard areas at the base of the cusps and along the margins; the mitral opening was rigid and a scant 2 fingers wide. There is atheroma of the aortic valves and arch with calcified plaques immediately above the aortic valve. The coronary arteries are thickened and sclerosed. The myocardium shows some fibrosis and there is a recent infarct at the lower interventricular area. There is no apparent pericarditis.

Abdominal Cavity

There are widespread fibrous adhesions throughout the cavity binding the viscera. The spleen and liver are so solidly adherent as to appear as one organ and have to be dissected apart. The spleen and liver are everywhere solidly adherent to the diaphragm and the capsule tears away in separation from the diaphragm.

Microscopic Findings

Heart — Sections show areas of fibrosis and some more recent degeneration. Blood vessel walls are thickened and arterioscelerotic.

Brain — **Cerebellar Tumor** — Sections show a rather vascular densely cellular tumor growth. The blood vessels are covered by endothelium only and surrounded by a pink staining zone around which the tumor cells are arranged like

collars. In other places there are pseudo-rosette, small circles of tumor cells with no central blood vessels. The tumor cells are elongated, oval with reticulated nuclei. Few mitotic figures are present. Ependymoma—epithelial type.

Ependymomas

Ependymomas comprise 3.7% of the gliomas. They occur chiefly in children in and about the ventricles. They grow slowly, but owing to their location, are not usually compatible with long life. Calcification within the tumor is quite common.

Although these tumors may occupy any part of the ventricular system, they are far more common in the fourth ventricle. Even though they occur more frequently in children, the age limitations are not nearly as sharply defined as in the case of the medulloblastomas.

Grossly the tumors usually arise from the ventricular wall on one side, fill the ventricle and push aside the wall and underlying brain tissue on the other. Thus they are often readily separated from brain tissue on three sides. In the fourth ventricle, they almost invariably arise from the floor or lateral walls, pushing upward the cerebellum to which they are not attached. On section they are rather tough greyish tumors.

Microscopically they are composed chiefly of two cell types: ependymal cells, and ependymoblasts. They vary in proportion in different tumors. The former is a polyhedral cell with a moderate amount of cystoplasm and usually fitted tightly together in a mosaic pattern. The ependymoblast on the other hand is a spindle-shaped cell with a rather long, fine process terminating on a blood vessel wall. The cells form a corona about each vessel, giving a typical appearance to the tissue. Both of these cells are characterized by the presence of small round or red-shaped bodies within the cytoplasm known as blepharoplastin. Not infrequently, cells arranged about a clear space similar to the central canal of the spinal cord, are seen in these tumors. This formation, known as a true rosette, is characteristic of the neuro-epitheliomas and is formed by the closely related primitive spongioblasts. Blood vessels are relatively numerous and the connective tissue is limited to their walls. Histologically, it is necessary to separate these tumors from the astroblastomas. Under low magnification, there is some similarity between the two, but on closer examination many differences become apparent. Blepharoplasten, typical of ependymal cells are never found in astroblasts. Astroblasts are much larger and their processes thicker and coarser. Ependymomas tend to be much more cellular and intervascular degeneration so common in astroblastomas, is rarely extensive in these tumors. Connective tissue hypertrophy so typical about the blood vessels of an astroblastoma, rarely occurs in an ependymoma.

Intra Cranial Tumor

Definition

The term "intracranial tumor" is conveniently applied to localized intracranial lesions, whether of neoplastic or chronic inflammatory origin, which by occupying space within the skull tend to cause a rise in intracranial pressure.

Etiology

The etiology of brain tumors is as obscure as the problem of tumors in general. We know very little concerning the factors associated with the formation of neoplasm. Biologically the only difference between the tumor problem in general and the specific problem of brain tumors is the phenomenon of metastases. The brain tumors, even gliomas, do not form distant metastases. The only metastasizing gliomas arise from the retina.

Tumors appear in all races and in either sex. There is a high incidence in childhood reaching a peak at about eight years. Another peak is reached in the middle of the fourth and another in the fifth decade. The most common age is around the years 45-50 followed by a rapid decline in frequency in the advanced years.

Heredity aparently plays a minor role in the etiology of brain tumors. Occasionally neurofibromata within the cranial cavity are found in more than one member of the same family. Cerebellar hemorrhagic blastomas are often familial in nature.

In a minority of cases, congenital abnormality appears to play an important part in causation, especially in the angiomatous malformations, the ganglioneuromas, the cholesteatomas, and tumors of the cranio-pharyngeal pouch.

There is no evidence that trauma is a predisposing factor, except rarely in the case of meningiomas which have been known to arise beneath the site of a previous head injury.

Frequency of Types of Intracranial Tumors Grinker	Frequency in 699 Cases	Average Survival in months (Cushing, 1932)	Longest Postop. Survival in Years
Glioblastoma multiforme	30.2%	12	4
Medulloblastoma	12.6%	15	7+
Astrocytoma	37.0%	16+	13
Astroblastoma	5.1%	37+	12+
Oligodendroglioma	4.0%	66+	15+
Spongioblastoma (polare)	4.7%	46+	10+
Ependymoma	3.7%	25+	—
Pinealoma	2.0%	18+	—
Ganglioneuroma	0.4%	—	—
Neuro-epithelioma	0.3%	—	—

Frequency of Tumors in Various Locations

Supratentorial—70%

Frontal lobe	16.8	Pituitary gland	2.7
Temporal lobe	13.6	Corpus callosum	11.8
Parietal lobe	11.8	Midbrain	0.9
Occipital lobe	2.7	Third ventricle	1.3
Anterior fossa	5.5	Multiple	2.3

Infratentorial — 30%

Cerebellum ..... 12.7 Fourth ventricle 6.8
Cerebello-pontine angle 10.4

Pathological Physiology

The functions of the brain depend on the maintenance of the circulation of the blood and of the cerebrospinal fluid at their appropriate pressures. The brain is unique among the viscera in being confined within a rigid box, the cranium. It follows that the total volume of the intracranial contents, the brain and its coverings, the blood vessels and the blood, and the cerebro-spinal fluid is constant, and that an increase in the volume of any one of them can only occur at the expense of the others. The intra-cranial contents, however, do not respond passively to changes in their volume or pressure, but react in complicated ways, so that any such alteration has far reaching consequences.

The increase in intracranial tension may, theoretically, be precluded by four possible factors: (1) obstruction of the ventricular system so as to prevent the out flow of fluid; (2) reduction in the size of intracranial cavity by an expanding lesion; (3) obstruction in the absorbing mechanisms, and (4) obstruction of the venous system draining the brain. The last two of these are, in all probability, rarely factors with intracranial tumors. Increased tension results in the majority of cases, from obstruction of some part of the ventricular system.

Clinical Features

A. Generalized Symptoms:

The presence of a tumor may give rise to early effects mechanically either through displacement of brain tissue or by mild block in cerebro-spinal fluid circulation.

1. Headache is commonly present, and is intensified or precipitated by those measures which tend to raise the intracranial cerebrospinal fluid pressure, such as stooping, straining or exercising. Conversely measures that reduce intracranial cerebrospinal fluid pressure may give relief of headache.

2. Nausea and vomiting are commonly manifested and not necessarily related to meals.

3. Mental clouding, lethargy and fatiguability are not unusual.

4. Generalized epileptiform convulsions are common manifestations of intracranial tumors. Walker found such attacks were the first symptom which the patient developed in 18% of all intracranial tumors.

5. Papilledema — as the intracranial cerebrospinal fluid pressure increases, papilledema occurs.

B. Focal Signs and Symptoms:

As the tumor grows, progressively greater destruction or dysfunction of tissue may occur, causing locally referable signs. Thus involvement of cerebrum brain stem, cranial nerves, etc., may soon be evident through the loss of function or altered function of these parts.

Accessory Methods of Investigation

A. X-ray of Skull:

1. Shift of a calcified Pineal gland may be present.

2. Local erosions or hypercalcification are sometimes present.

3. Ballooning of the sella turcica is sometimes evident.

4. Following prolonged increased cerebrospinal fluid pressure of at least six months duration, digital markings of the inner table of the skull may be present.

5. In children abnormal separations of the sutures may be present.

B. Lumbar Puncture:

Increased cerebrospinal fluid pressure (over 200 mm. water) and increase in protein are apt to be present. Lumbar puncture should be carried out with caution in suspected cases of intracranial tumor, especially when there is reason to suspect that the tumor is in the posterior fossa.

C. Visual Field Examinations:

Involvement of the optic nerve, optic tract or radiations may give rise to classical visual field defects.

D. Ventriculography:

1. Air introduced directly into the ventricles may indicate obstruction, deformity, dilatation, shift and other changes of the ventricular system.

2. Air encephalography (air introduced via lumbar subarachnoid spaces) is ordinarily considered contraindicated when a strong suspicion exists for the presence of an intracranial neoplasm.

E. Electroencephalography:

The presence of focal abnormalities, especially a delta focus, is frequently associated with the presence of cerebral neoplasms.

F. Arteriography:

The injection of diodrast or thorotrast into the internal carotid artery may show displacement or obliteration of major vessels or outline the presence of a neoplasm.

Treatment and Prognosis

Prognosis varies with the type and location of the individual tumor.

Cure may be effected in some tumors such as maningiomas, neuranomas, dermoids and astrocytomas by early diagnosis and removal of the neoplasm.

On the other hand, a deep seated tumor such as glioblastoma multiforme, medulloblastoma and ependymoma may be entirely unaffected and even unfavorably influenced by operation.

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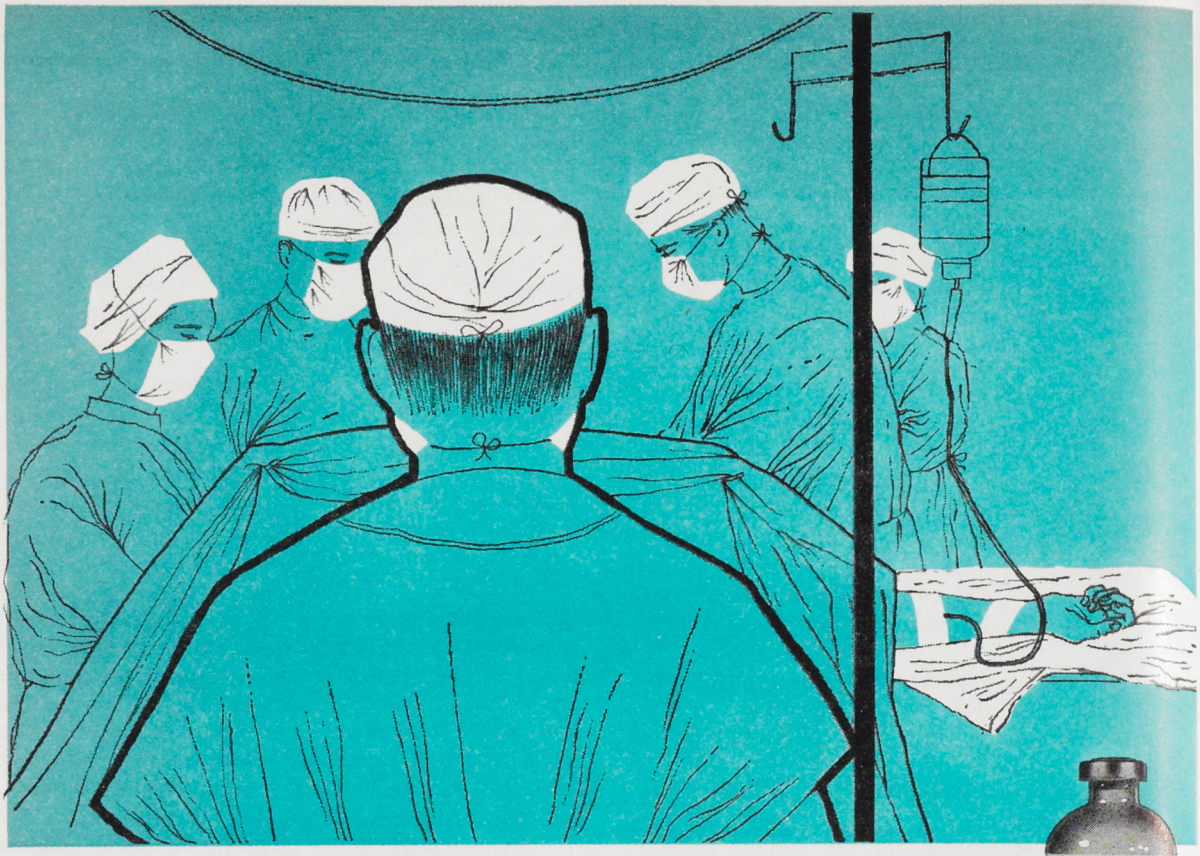

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WALKERVILLE, ONTARIO

Lecture

Research and the General Hospital

A. J. Glazebrook, M.D., London
St. Boniface Hospital

Mr. Chairman, Ladies and Gentlemen:

I wish to talk tonight about medical research in a General Hospital, and it might be useful to consider this subject from two points of view. Firstly, the importance of medical research, and the obligations a general hospital might feel impelled to assume because of this, and secondly, the administrative difficulties which might arise as a result of efforts to set up a research department in a hospital.

We could best consider the importance of research from the standpoint of the individual doctor, for after all a hospital represents a medical workshop, and the combined efforts of a number of doctors. That which is of great concern to the individual must affect all of the workers in the group.

We could look at this problem purely from the economic angle, and in Great Britain where the National Health Service costs something like 12 hundred million dollars a year, the bill is one that must be shared by every citizen and makes up a large part of the crushing taxation which these people bear, a taxation that a short time ago seemed likely to kill all enterprise and endeavour, and slowly asphyxiate this island race.

No one in Great Britain is more aware of the cost of the National Health Service, and the great difficulty of confining it to the 12 hundred million dollar limit, than the doctor; and every day it is borne in upon him; the loss of man hours, the loss of production; the increase in hospitalization charges, and the staggering drug bill; because he has to pay his share of it and he sees the money being spent.

Things are really no different here, where the citizen and not the State, pays the medical bill. The money still has to come from the production of this country; from the wheatfields, and from the mineral deposits; from the farmers, and from the industrial workers.

Think for a moment of the economic value to the people of the discovery of the antibiotics and the sulphonamides, substances which have changed the course of so many diseases, to an extent almost unbelievable, so that their very nature has changed; from being harbingers of death they are now nothing more than incidental illnesses.

Twenty-five years ago when I was a medical student, I used to walk round the wards of a London Hospital with my Chief and examine with him young people sick with lobar pneumonia; and he would shake his head, and say this one will die, and that one will perhaps live, and that was all he could do; for there were no antibiotics and no sulphonamides.

Lobar pneumonia is no longer a killer. No longer does it pluck the young man from the threshold of life and love. Medical Research has seen to that, but there are less dramatic diseases which cost as much in treasure.

Peptic ulcer, for instance, still challenges the best brains of medicine. It affects the working population of every modern community; slowly sapping the energy, the initiative, and the skill of the bread winner; so that at a time when his experience and knowledge have ripened into a valuable asset to his country, he falls a prey to the canker gnawing at his vitals.

In Denmark the incidence of peptic ulcer amongst males increased by 300% during the period 1940 to 1948, and at present it afflicts over 6% of all males there between the ages of 40 and 55.[1]

In Great Britain 9.6% of all men in this age group have the disease, and I doubt whether its malevolent influence is felt any less in the North American Continent.

Besides the economic or bread and butter factor there is a moral or ethical reason which must stimulate the doctor to research; the fact that he is bound to do the best he can for sick people, and explore every possible avenue to effect a cure; where this is unknown he must strive to find the answer, and by patient observation and deduction endeavour to add to human knowledge.

Finally there is a personal reason. The doctor is not immune to disease, neither is his family. He sees it in its most horrible and terrifying forms; the intolerable pain that attacks the integrity of the mind during the long night; blindness, paralysis, mental disease; the eroding cancer which can change the loved one into a monstrous shape of phantasy.

Last year when I came to Winnipeg, I went to the Municipal Hospital and saw the young people in respirators; here a child looking at a doll she could not hold, there a young man gazing at the picture of the girl he would never marry. Supposing it was my little girl, I thought: condemned to slow death in this fashion, cruelly confined in this way, looking at me for help, her back covered with bed sores, her arms and legs wasted to match sticks; what would I do? what can we do about this affliction?

From a lecture given to the Extension Course in Hospital Administration organized by the Canadian Hospital Association at the University of Manitoba, July 9th, 1954.

No doctor could question the importance of research on any one of the grounds that I have mentioned, or fail to seize the opportunity to push forward research projects if within his power; as with individual doctors, so with the groups working in hospitals; where the opportunity is so much greater, and the sharing of knowledge and experience facilitates progress. So far I have spoken of economic, ethical and personal reasons for promoting research as though they concerned only the medical profession, but all of the workers in the hospital, and indeed everybody in the community, is under the sanction of these same reasons if only they knew it.

An active research unit does not do a hospital any harm. It seems to stimulate the general standard of work; it gets the best out of people; as a hospital becomes known by the publication of good papers, the staff, both lay and medical becomes increasingly proud of their Institution; visitors from near and far are attracted; what was before competent or mediocre now becomes first class, else there is nothing to show; the Hospital can afford to become more choosy in selecting new staff and so healthy development is accelerated.

There is something else worth mentioning. I do not want to enter into controversial matters, but I believe that for political reasons some measure of State Medicine may become inevitable in all civilized communities, although not necessarily after the British pattern.

In Britain, Hospital Management Committees were frequently set up in one of two hospitals, the well known hospital absorbing the unknown one, which then proceeded to lose its identity and autonomy. Although it perhaps had been doing excellent work of its kind in the hands of busy people, too busy to notice the march of events until too late; the lamentations and petitions afterwards fell on the stony soil of Governmental indifference. In other words, active research units might aid a hospital in the maintenance of its independence and dignity under such circumstances.

Unfortunately, the setting up of a research unit in a hospital offers administrative difficulties, and many people, while they are extremely enthusiastic, lack insight into the problems. Research is to them a wonderful dream; they think in terms of boffins or back room boys shaking test tubes in some hidden corner and producing miracles. They do not see the blood, the sweat, and the tears which are required to produce results.

Research in a hospital should be essentially clinical and based on a close study of sick people. The test tubes, and the boffins are better left in the hands of the pharmaceutical companies, who have the resources and the ability to outstrip hospitals in this sort of thing. What these companies would give their souls for, however, are the clinical facilities of the hospitals.

The efforts of the research department to acquire clinical material, while not directly an administrative problem, will set up stresses and strains which the administration is bound to become aware of, and it may be looked to for guidance. While Staff men are anxious to assist, they are leery of handing over patients to a director who may be unknown to them. Later he may compete against some of them for private patients and this may arouse fears and suspicions.

Patients themselves are not always anxious to enter hospital under the care of a director of research, as some do not like being guinea pigs. In Britain this situation is met by making the Clinical Professors responsible for research; and the title of Professor, an uncommon one there, attracts patients rather than the reverse.

A research director is perhaps better paid on a full time salaried basis with private practice prohibited; this would remove some of the fears based on possible competition. Unfortunately the able men in this country must strive in fields where they obtain maximal financial rewards; and research resources are often too meagre to permit good salaries and to attract able men, so some supplementation of their income by private work should be allowed.

In Britain the position is different. There the Exchequer is painfully aware of the burden of the Health Service; knows that medical education and research offer the only practicable alleviation; and realizes that fame is not the only spur, and that a place in the sun may be a more powerful inducement. Thus the system of merit awards has been introduced, by which the brilliant educator and research worker may earn nearly twice as much as the competent surgeon or internist. This excellent scheme has been sadly weakened and made suspect by the conditions of secrecy which surround the issue of merit awards, for publication of the names of the recipients is not allowed.

A research department must have hospital beds for basic studies; but the word research should not be associated with them; they should be called metabolic units or clinical investigation wards. The nursing staff attending these patients must be alive to the importance of adhering strictly to instructions, and the great loss of time and labour which will result if they are ignored.

Some laboratory space must be set aside for special techniques and procedures. It is better to have an entirely separate laboratory; for if the work is carried through in routine labs, it will suffer when there is a heavy rush. This is a council of perfection, and might prove impracticable for reasons of space and the heavy cost of reduplicating equipment.

The research must not be allowed to interfere in any way with the comfort and well being of the patients, and their investigation and treatments must take first place; neither must research conflict with the existing moral standards of the hospital.

An administrative problem in the early days lies in the control of research funds and procedures. Research funds can be obtained from many sources, but great care should be taken to see they are spent only for the specific project for which they have been supplied. There is a great tendency to regard these funds as expendable for all sorts of things, and if they are improperly used damage will result.

And there is one last thing. If you have a British doctor as a director, take pity on him, take him on one side and explain to him the vast differences which exist between British and North American Hospitals, in case he sinks in a sea of bewilderment and does not come up for a third time.

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Obituaries

Dr. Isaac Pearlman

Dr. Isaac Pearlman, 68, died in St. Boniface Hospital on August 4. Born in Russia, he came to Winnipeg in 1911, graduated from Manitoba Medical College in 1918 and practised continuously in north Winnipeg. He served on the staff of St. Boniface Hospital in charge of gastro-enterology and was a lecturer in internal medicine on the medical faculty of the University of Manitoba. He served as president of the College of Physicians and Surgeons of Manitoba and was made a life member of the Winnipeg Medical Society.

In addition to having a keen interest in music, he served as a member of the Winnipeg Public Parks Board, was president of the Simon Flexner club, a founder of the I.T. Peretz Folk Schools of Winnipeg and a leading member of the Canadian Jewish Congress, Western division.

He is survived by his wife and two sons, one of whom, Dr. Leonard A. Pearlman, graduated from the University of Manitoba in 1953.

John Cruikshank Hossack

Dr. John Hossack died on August 16, aged 63. The manner of his death was so sudden that few knew of it until a day or more had elapsed; when realization came it was evident that a unique personality had passed.

Perhaps it was inevitable that a lad born in Macduff, Banffshire, Scotland, should be a student of Shakespeare and indeed of all good literature. It was characteristic of John Hossack that the latest request he made of the medical librarian was for a book, the Bible and Modern Medicine, though before he could call for it his summons had come. It was also characteristic that only a few weeks ago he drove out to the University to hear a paper by Dr. W. A. Scott of Toronto, on "Medicine in Ancient Rome" read before the Classical Association of Canada.

His two loves were medicine and literature. His mastery of medicine is attested by his becoming a demonstrator in medicine in 1925, four years after graduation, and progressing to become assistant professor in 1939, by the interest which successive classes of students took in his clinical lectures and by being frequently sought by his confreres as a consultant in obscure cases especially those with a neurological basis. An indication of the respect in which he was held in his profession is that he was elected President of the Winnipeg Medical Society, later a life member of that body, and Honorary President of the Manitoba Medical Students' Association.

Literature was almost an even competitor with medicine in his affections. The Dean of Law stated that for years he and Hossack had been members of a literary society which met monthly. He was the moving spirit in the formation and continuance of the Medical History section of the Winnipeg Medical Society and he contributed many articles to medical journals. From 1943 to the time of his death he was editor of the Manitoba Medical Review and was largely responsible for bringing it to its present proud position. From his wide reading, especially of the older authors, he developed a classical style of speaking and writing which on occasion enabled him to rise to the heights.

For all his knowledge of books he was not bound by conventionalities but radiated his own individuality.

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Editorial

Manitoba's Medical Men

X. Conventions

The Canadian Medical Association had their annual convention in Vancouver in June and it was one of the largest conventions on record. The president who worked indefatigably for eighteen months is one of Manitoba's Sons. His reward after sacrificing time, practice and himself, is the satisfaction of knowing that he served his colleagues well.

The most outstanding event of the convention was the establishing of the Canadian College of General Practice. The first president of this newly formed association was presented with a gavel carved from a 3,500 year old tree under which Hippocrates studied medicine.

The convention of the Manitoba Medical Association will be held in October of this year and attendance is usually very good with the interest of the doctors high. All the committees and sub-committees of the executive and those appointed by the president are working industriously to make this convention outstanding. There will be several visiting speakers on the scientific programme as well as many panel discussions.

Here in Manitoba the displays of the pharmaceutical and surgical supply houses are very well attended by the doctors. This is important because part of the expense of putting on the conventions comes from these sources.

Doctors are reminded once again that early registration is important and that the Federal government allows the amount incurred for these conventions as an income tax deductible expense.

The Public Relations Committee is very anxious to receive copies of the scientific papers which are going to be presented, not only for the Manitoba Medical Review but also for the newspapers.

L. A. Sigurdson, M.D.

Letter to the Editor

827 Cherry St.,
New Westminster, B.C.,
June 29th, 1954.

The Editor,
Manitoba Medical Review,
604 Medical Arts Bldg.,
Winnipeg, Man.

Dear Sir:

My purpose in writing is to appeal to the medical profession to take action against the terrible threat of mass radiation facing all people. The possibilities for good or evil inherent in the liberation of nuclear energy are apparent of course, but which is to take precedence is still undecided.

There is a tendency, in some circles, to blame the scientists for the impasse which humanity now faces. However, the record shows that from the beginning, the great majority of scientists, including those working on the bomb were strongly opposed to the use of atomic energy for destructive purposes. For example, the Franck report (June, 1945) presented to the U. S. Secretary of War, argued cogently that the use of such weapons, would be immoral, unnecessary, alienate world opinion and precipitate a nuclear armaments race. Recently we have the fate of Dr. Oppenheimer, apparently for opposing the H-bomb, as further evidence.

United action against the bombs is difficult in the U.S.A. just now because of McCarthyism, but individual scientists have nevertheless made important statements on the subject. Dr. Albert W. Bellamy, University of California professor of biophysics stated on May 8th, "We have not lived long enough with radiation to know yet just how much long continued, low level radiation — both internal and external — we can live with without injury . . . " Dr. Gordon Fitzgerald and Dr. John N. Heslep of the above University also made statements on the danger of radioactivity. Drs. Dade W. Moeller, James G. Terrill and Samuel I. Ingrahm II (Pub. Health Rep. 68, 57-65, January, 1953) pointed out the danger to this continent of uranium mining and milling, nuclear reactor operations, and testing of weapons. In Britain, Prof. C. V. Powell, Nobel prize winner, and retiring president of the British Association of Scientific Workers declared recently, "Our situation is one of extreme gravity. One can almost hear the tolling of the great bell of history." The above association endorsed an executive committee statement on the H-bomb, welcoming the world reaction against the recent tests. The statement also said "Scientists have a special responsibility to give a clear warning to the public on the implication of recent developments." Prof. Powell also stated, "It is our duty repeatedly to emphasize that control (of nuclear weapons) is technically feasible; that all nations are agreed on its necessity; that both the U.S. and the U.S.S.R. have accepted the principle of permanent inspection and of a system of control, not subject to veto, whereby suspected violations may be investigated." Without such control the whole world will continue to live in the shadow of a frightful catastrophe."

You may be aware that twenty professional people in the greater Vancouver area, three dentists and seventeen physicians including two radiologists sent a letter to the Prime Minister warning of the dangers of radioactivity and requesting that Canada take the lead in calling for the abolition of nuclear weapons.

The governments of Japan and India have called for the banning of atomic tests and weapons and in our own country the government of Saskatchewan did the same. The B.C. legislature also showed their strong feelings in this regard.

Despite the above and numerous other reactions, H-bomb tests were continued, the last three secretly, presumably because of public opinion. We have now been promised further tests both in the Pacific and on this continent. As well as this the Dulles policy of "instant massive retaliation" has not been repudiated. Under this policy Korea would have been the end of all of us. More protest is evidently needed. As this continent is one of the main keys to the problem and as the atmosphere in the U.S.A. is not conducive to articulate protest it becomes imperative that Canada, especially its scientists, give a lead. Medical men's claim to leadership, especially in the health field, cannot survive the ignoring of this problem. Also, our own existence is at stake.

According to "Life" magazine (April 12) the radioactive cloud of the March 1st H-bomb was 150 miles in diameter as compared to 2 - 3 miles for the A-bomb. We have radioactive ocean, 3,000 miles either side of Bikini, radioactive rain in Japan, "well above the danger point," radioactive dust on the coast here "just below the danger point." On May 17th, Dr. Lincoln Lapaz, head of the University of New Mexico department of meteoritics stated that background radiation had been up to 35 times normal for five days. Reports from other places, notably Wyoming and Utah showed similar jumps in radioactivity.

I have no doubt that nuclear weapons will be abolished, the sooner they are the less damage will be done. Also, physicians, concerned as we are with the preservation of life and health should be in the forefront.

I may be "carrying coals to Newcastle" in this letter but at least it can do no harm. Resolutions from medical groups or even individuals calling for the end of nuclear weapons and their testing and released to the press could have a great beneficial effect.

Yours sincerely,

F. A. Walton, M.D.

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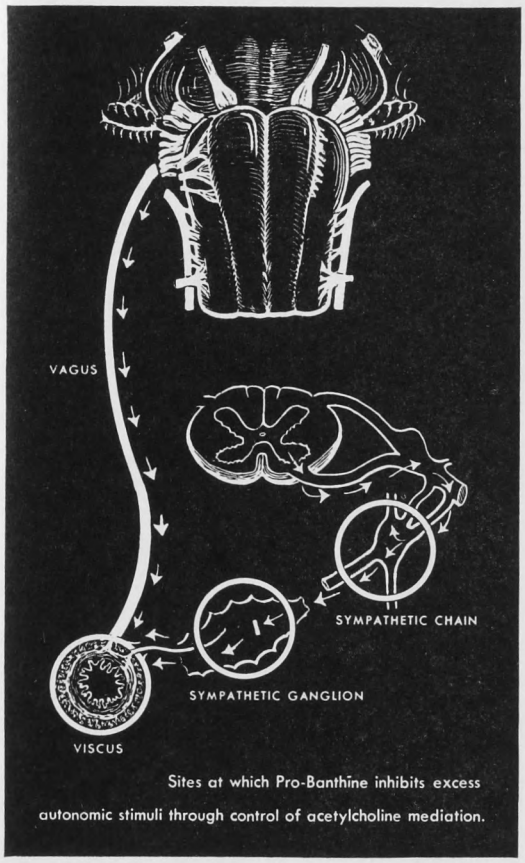

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Dr. Isaac Pearlman — An Appreciation

Isaac Pearlman

Isaac Pearlman died on the 4th of August and our local profession is the poorer by his death.

He was in many ways a remarkable man. A Jew by race, a Russian by birth, his youth was spent in an atmosphere of persecution and unrest. "Where there is love of man there is love of the Art" is an aphorism of Hippocrates. In Pearlman's heart there was much love of man and so he turned to the Art. At that time there were in Russia different grades of medical training, and the one best suited to his purse might be regarded as comparable to that at one time given by the Society of Apothecaries.

He was successful in practice but was so deeply moved by the sufferings of the poor and of his own people that he became interested in politics — a most dangerous interest in Tsarist Russia. His activities came to the notice of the government and, as he stood in danger of arrest, he was urged to flee. He did not dare apply for a passport in his own person or in his own name but a relative, whose name was Pearlman, secured one for him and on this he was able to secure passage for Canada where he arrived in 1911. The immigration authorities noticed discrepancies in his papers and, in explanation, Pearlman, as he was now called, laid before them his whole story. The officers listened and were satisfied; and, with the consideration and sympathy that British officials have always shown towards the distressed, they gave him asylum.

He came to Winnipeg friendless and almost completely ignorant of the English language. To repair this latter defect he engaged a tutor at twenty-five cents an evening but, in his own words, he "wasted" only fifty cents on this instruction. Instead, he listened, spoke and read, and soon had gained an understanding, if not a mastery, of his new tongue. In time he spoke with fluency and clarity but to the end he retained an accent and phraseology that gave piquancy and flavour to his speech and held his audiences.

His training was not sufficient for him to obtain a licence to practice, but a busy co-religionist whose patients were mostly Jewish and Slavic, gave him employment as assistant. This supplied him with occupation and income, but he had no intention of remaining on the fringe of the profession. Accordingly, in 1913 he approached the College authorities and was told that he would be admitted to the Second Year provided that he also took the course in Anatomy. He made a satisfactory pass even though his hours for study were cut into by the duties of his assistantship which he had not relinquished. These duties and Second Year Anatomy made much heavier the already heavy Third Year Course; yet, at the end of the year he passed, not only with honours but with a scholarship, a remarkable achievement.

At that time the Professor of Physiology was Swale Vincent, the distinguished endocrinologist. Pearlman's work as a student attracted Professor Vincent's attention and led to the offer of a position in his department. For Pearlman it meant the interruption of his course but it meant, also, congenial work and, not least, an escape from his "practice." During the year that he held this appointment he actively assisted Professor Vincent in his investigations and was instrumental in proving that the cutaneous vasoconstriction produced by adrenalin was accompanied by myovascular dilatation. He made certain not unimportant discoveries of his own in this new field and Vincent spoke highly of his work.

His appointment had been for a year only and Professor Vincent was anxious to renew it, but Pearlman was equally anxious to obtain his degree as quickly as possible.

Largely through the good offices of Professor Vincent it was arranged that if he could pass the Fourth Year examinations in September the College would not be critical of how he prepared for them. He therefore took a course in Chicago, acquitted himself honourably at the fall examinations and rejoined his class for the final year. He still gave occasional assistance to Professor Vincent and even to his original chief, for these extra-curricular activities kept him in funds.

Pearlman was one in whom Maimonides would have rejoiced. His eye was keen, his mind was alert but his heart was sympathetic and his disposition was kindly. He had the gift of empathy and a rich understanding. As is, and has been, true of every good physician of every age and clime, it was the patient rather than the disease that held his chief attention. Thus he practiced psychosomatic medicine long before that term had been coined.

In Chicago one of his teachers was Sippy and the attitude and enthusiasm of this great teacher so inspired Pearlman that from then on his chief interest was gastro-enterology. On that subject he was recognized to be an authority. He observed carefully, read critically and reasoned clearly. The theories and practices of the moment left him untouched save where they appealed to his reason or agreed with his experience, and because of the soundness and saneness of his opinions he was listened to with respect and his advice was followed with confidence.

His colleagues found many ways to honour him. He was repeatedly elected to the Council of

the College of Physicians and Surgeons and was for a time president of that body. He was given honorary life membership in the Winnipeg Medical Society. Because of the excellence of his instruction he was retained as lecturer in medicine after th usual age of retirement, and when he resigned from the staff of St. Boniface Hospital he was appointed an Honorary Consultant.

Isaac Pearlman's kindliness and skill attracted to him a large practice in which were the very wealthy as well as the very poor. If his attitude towards these extremes differed in any way it was only because he found his greatest satisfaction in serving those from whom he could expect little in return but gratitude.

He had many interests. The sufferings of his people concerned him deeply and he was active in all the local movements that had as their purpose the relief and re-establishment of those unfortunates. He was a chess enthusiast. He was keenly appreciative of music, and his evenings at home were made particularly happy by the brilliant playing of his wife and son. These and his other interests gave variety to his life and kept him youthful. He was fortunate in his affections and his affairs and, most of all, in that he was not called upon to endure that death-in-life which is the unhappy lot of those who still must live after their flame lacks oil.

He will be missed by very many—his friends, his colleagues, and most of all, his patients. In all of these his memory will be slow to die, and by all he will be pleasantly remembered, for he had a good name, and, in the words of an ancient writer of his ancient race, a good name is more to be desired than great riches.

Victorian Order of Nurses

Today, with the rising cost of medical care, especially hospital services, and the need to maintain our voluntary system of medical practice, it is more important than ever for physicians to utilize visiting nursing service. Better understanding of the visiting nurse's contribution to medical practice is the first step towards its more effective use.

The service is organized and administered by a group of public spirited citizens—the local Board of Management. A medical advisory committee guides the agency in its professional services. This guidance includes preparation of standing orders to be used when a nurse is called to give emergency care to a patient who is not yet under the care of a physician. Nursing care cannot be continued to a patient who does not have medical supervision. Nurses are instructed to keep in close communication with the attending physician on each case.

Contrary to the belief of many lay and professional persons, visiting nursing is available to people of all economic levels (just as hospital care is available to all) and is **not** limited to service to the indigent. Cost of the service is carefully computed each year. In Winnipeg the present cost of a nursing visit is $2.00. Those who can pay for their care are expected to do so. Reduction or waiver of fee is arranged when necessary by the nurse who cares for the patient. She does this after discussion with patient or family. Last year the full fee ($2.00) was received for 14% of the visits made by the nurses. Part-pay which varies from 25 cents to $1.75 was received for 43% and 43% were free. Care provided on a free or part-pay basis is made possible by various grants, the largest of which is from the Community Chest.

The Victorian Order has an adequate staff (all registered nurses) to care for your patients in their homes. Medical, surgical, convalescent, and maternity patients can be cared for. Telephone 92-8529.

Article

Ave Atque Vale

E. Moorhead, M.B.

It is one of the regrets of elders that they cannot pass on to the next generation many of the experiences that have been acquired by time, observation, trial and error. Techniques can be illustrated and explained, but these deal mainly with methods and do not solve major problems.

Lecturing and teaching came to us from an age when books were so expensive or so difficult to come by that they were beyond the reach of the average student. These two requisites were dealt with in a series of articles in "The British Medical Journal" two or three years ago, and are worth serious study by deans and those who assist in making appointments to the teaching staff. The usual course in appointing professors or lecturers is to choose one who is skilled in the practice of the subjects which are to be taught. That he can lecture or teach may never be considered. For the former a course in elocution is advised, since many a potentially good lecture or address may be spoiled by a poor delivery or inadequate preparation. A young man is likely to devote more time and thought to a subject than his senior.

Can he teach? Test it for yourselves. Ask someone who has been to an instructive address, what he thought of it. "Oh, it was very fine and most interesting." "But what was it all about?" "Well, I found it very hard to follow and I cannot really explain it all to you."

Never apologize for your address. To my mind, the most insulting opening by the speaker is to tell his audience that he had not time to prepare his subject, but had hastily jotted down a few notes. I have never dared but would like to tell such a one that since we had paid him the compliment of coming to hear him, the least he could do was to render us the courtesy of careful preparation.

Many speakers fail to observe their audiences. If they did, they would find that they had lost their attention. The restlessness, coughing and whispering are ignored, and the speech instead of being wound up, drags on to a dreary end.

It is often forgotten that while the doctor is sizing up the patient, the latter is doing the same to him. I had gone away for a few days, and when I returned the mother of a five-year-old girl gave me an interesting story. I had left the care of the child to a well-known practitioner. After leaving the bedside he asked the mother why the little girl said, "Do you wish to wash your hands?" several times. The mother explained that when I had finished my examination, I always asked permission to wash my hands—a British custom of fifty years ago which has stayed with me. The child was intimating quite politely that she wished he would go away.

Our dealing with children frequently shows a lack of understanding, and is a reason why children often dread the doctor's visit. An abscess has to be opened or a needle inserted; mother and doctor join in a chorus to tell the patient to be good, that it will be over in a minute, and won't hurt a bit or only a little. A small boy on Wellington Crescent had fallen out of a tree, laid his cheek open, and would require three stitches. Father and Mother struggled wordily to minimize the operation, and Tommy knew it. After getting things ready I told him that it was poppycock to say that it wouldn't hurt, but I had a rule that I wouldn't have a boy held down while I was doing the stitching. "Suppose we send Father and Mother off for a ten-minute walk, and you and I will finish it between us." He enjoyed telling the story that he did not cry or struggle, and later when I told him that taking the stitches out wouldn't hurt, he believed me.

A well-known Dublin surgeon, Sir Thomas Myles, used to correct a student who had elicited a yelp of pain from a patient whom he was examining. "Don't hurt the patient; anyone can do that. A doctor ought to be able to examine him without hurting him." I had much difficulty in driving home one point with my internes—Don't look at your hands; look at the patient's face especially if there is pain in the abdominal area. A doctor should never have to ask, "Does that hurt?" If he is watching the face, he will note the flicker which denotes that he is approaching a painful region and he will proceed very cautiously, otherwise the muscles will harden to protect the underlying organ and further examination will be painful and of little value. Just note how many of your medical friends look at their hands.

We have made wonderful progress and learned many new things in the last fifty years, but have overlooked some of the valuable assets of those days—one in particular, the cultivation of observation and reasoning. Like the rest of the world, we place much of our trust in technical assistance, even in such a commonplace thing as a thermometer. If there is something in the picture that is missing, suspect your diagnosis and revise it.

A patient had already been in the ward for six weeks with a diagnosis of sub-acute bacterial endocarditis, running an irregular temperature, sometimes very high and never returning to normal. Our examinations gave no positive evidence of endocarditis. During a quiet period I had him sit up in bed and the temperature shot

up. A few days later I instructed my interne to get him out of bed on to the floor and note carefully what happened. Next morning a very scared interne reported that the patient had collapsed on the floor and looked as if he would pass out. "What was his colour?" "No change." "His pulse?" "Normal." "Young man, you are letting your imagination run away with you, pinning your faith to a thermometer, and ignoring a pulse rate which never increased with the high fever." Next morning an unexpected early return of the nurse who took the temperatures found the thermometer reading 110. There had not been time to shake the mercury down to a more reasonable figure. A radiator or fomentation had provided the fever.

A common telephone conversation in my experience—"Good morning, Mrs. Blank. How is Johnny this morning?" "Oh, Doctor, his temperature is 101." "I did not ask what his temperature was; I asked how Johnny was." "He had a good night, Doctor, and he wants some breakfast."

No one would deny the great value of radiology. But the use or rather misuse of it has tended to give some radiologists swelled head, make the public believe that we see all the internal organs and cultivate in many practitioners a "laisser faire" attitude which does not require serious mental effort on their part and will provide the diagnosis not yet made. After all, you cannot tell a man's character from his photograph, nor differentiate between a screen star and a gentlemanly bank robber by their looks. Its real value lies in confirming a diagnosis, and we must remember that it has added little to the knowledge that Colles gave us of fractures of the wrist. If it fails to confirm, then survey the reasons by which you formed your opinion. No interne of mine was allowed to order an X-ray until he had arrived at a diagnosis based on history, physical findings, etc.

A patient gave evidence of an empyema after an attack of pneumonia, and an X-ray was ordered. The report was negative. In reply to the query of, "What next?" the interne could not answer except to say that evidently there was no empyema. "Then you sell your soul to the machine," was my reply. "We have worked hard on this case and our diagnosis is justified. You will try this afternoon with a syringe to find that abscess. You may have to try several places." Next morning he displayed a syringe full of pus, and asked what he was to do with it. "Send it to the X-ray department with our compliments, and tell them that they haven't got the last word." I think the interne will remember the incident.

I prefer the title Family Doctor to G.P., but suppose it would not differentiate the group from the specialists. It is a more friendly term, and the Family Doctor was often consulted on family affairs. In one large Roman Catholic family, the children frequently addressed me as "Father." To be classed in the same category as the parish priest was indeed a compliment. To be told, as one was not infrequently, "I felt better every time I heard your step on the stair," shows that there is something more in treatment than a diagnosis and a bottle of medicine. I am still told by former patients that my successors seem to be always in a hurry and never have time for the friendly chats which were enjoyed by the patient—and often gave information that would not be gained by direct questioning.

One thing should be remembered by the visiting doctor when the illness is long and is being treated in the home. You have been visiting on your rounds and usually come about ten o'clock. Some emergency upsets your routine, and a visit will not be possible before the afternoon. Always telephone of the change. Otherwise you are likely to find a set-back, higher temperature and pulse, headache, could not eat the usual meal, etc. The visit of the doctor is one of the most important events of the day for a sick person. Every time a car stopped, the front door bell rang, or another voice was heard downstairs — that must be the doctor. Disappointment, restlessness, etc., are the result of hope deferred. Never promise some increased activity, food, etc., ahead. Stall if you wish. In charge of a Dublin fever hospital over fifty years ago, I was recovering from typhoid fever and had been promised my clothes on a certain day. Owing to an oversight, the order was not transmitted. I could have howled with rage and vexation.

Several years ago a campaign, such as our neighbours to the south love, was instigated: diagnosis, diagnosis, to the nth degree — very worthy, but with little mention of curing the sick man or woman who was just a case of so and so. If you are honest with yourself, you will recognize that many of your patients recover on a very shaky diagnosis. After all, Christian Scientists cure a certain number of undiagnosed ailments, and you can do the same. It is a happy day when you are able to say, "I don't know," but don't try it when you are young; another doctor will be called.

A doctor told me that he could not examine a patient's chest because he had no stethoscope. I think that I was able to convey, without hurting his feelings, that by putting his ear against back or chest, with or without an intervening towel, he would learn just as much as with a stethoscope.

Intentionally I have not referred to the very wonderful progress that has been made in the last half century. But I would like to point out that some of our discoveries were known for many

years, though not understood. It was common practice, and I have seen it done by oldfashioned midwives and wise women, to carry out a certain ritual after a baby was born and washed. A square of linen was placed on a hot stove. It must be scorched. And then it was applied as a dressing over the umbilical cord. Those women were using asepsis long before Lord Lister's time. It is lucky that ironing of linen and cotton goods improves their appearance, for the housewife would never bother about it for the sterilizing benefits! Tribes and nations in many parts of the world know that certain kinds of mud or clay help the healing of wounds. Agents are now searching for the areas where these are to be found to add to the list of antibiotics. Tribes in South America have long used curare to assist in killing animals with spear or arrow. They were aware of the value of its paralyzing effects. Recently we have obtained assistance in major surgery by its use in moderate amounts. The dictum still stands: "What is good is not necessarily new."

How much should a patient be told is often the most difficult problem the doctor has to face. Many good Christians fear dissolution and wish it deferred regardless of pain. Others wish for it as a welcome relief from suffering. There is no rule I can give. To those who want the truth, don't hedge, but don't put a time limit. Doctors are poor prophets. To the timid you can only offer every service that may help without committing yourself. In the days of the typhoid epidemics half a century ago, I often came to a house expecting to see crepe on the door, and found that the patient had had a good night and seemed better. Those were mostly young people. For older people I have always chosen to smooth the passing rather than to harass with attentions. I hope that when my time comes, my doctor will remember that.

Urology Award

The American Urological Association offers an annual award of $1,000 (first prize of $500, second prize $300 and third prize $200) for essays on the result of some clinical or laboratory research in Urology. Competition shall be limited to urologists who have been graduated not more than ten years, and to men in training to become urologists.

The first prize essay will appear on the program of the forthcoming meeting of the American Urological Association, to be held at the Biltmore Hotel, Los Angeles, California, May 16-19, 1955.

For full particulars write the Executive Secretary, William P. Didusch, 1120 North Charles Street, Baltimore, Maryland. Essays must be in his hands before January 1, 1955.

Social News

Reported by K. Borthwick-Leslie, M.D.

To Dr. J. C. Hossack—and I am quite sure he is up there, watching, reading, listening and I bet criticizing—my sincere regrets for never getting over for that long promised session in the library and art gallery. To Mrs. Hossack and family my personal sympathy.

To the new editor whoever he may be my sympathy too—you have a small size shoe to fill, but a third dimension of culture, literary knowledge and ability to live up to.

♦

Through the summer so many went so far and to so many interesting places, space does not allow individual reports, so to all happy landings back at home and the after-math of all good holidays. In reverse, it is indeed a pleasure to welcome back to our fold Dr. Robert L. Cooke, who for some three years has been doing Cancer Surgery in the Memorial Centre for Cancer and Allied Diseases, New York. This year he became a Diplomate of the American Board of Surgery. He is now associated with the Medical College and General Hospital continuing his Cancer Surgical work.

♦

In June, Shirley Edna Swail, only daughter of Mr. and Mrs. Roy Swail, was married to Dr. Wm. Leslie Parker, only son of Mr. and Mrs. W. V. Parker, Twickenham, Middlesex, England. Mrs. Parker is a fifth year student, Dr. Parker a graduate of Edinburgh University. They will reside in Winnipeg.

♦

Speaking of the army, Dr. N. R. Rawson (ex Major) and at one time Geo. Wakefield's big boss, has retired from his position as medical director of St. James, St. Vital and Fort Garry health units after eight years of service. All told twenty-two years of service as a public health officer, including the tour of Epidemiologist in the R.C.A.M.C. The O.B.E. is because of fighting a Typhoid epidemic among the Esquimos in Baffin Island in 1945, where he also earned the name "Ishauruk"—(The little doctor). I understand Dr. Rawson, always alert and active wishes to remain so, as in doing Locum Tenens, etc.—so please remember.

♦

Congratulations to Dr. Alan A. Klass, B.A., M.D., F.R.C.S. (Edin.), F.R.C.S. (Can.), on his appointment as Chairman of the Winnipeg Men's Branch of the Canadian Institute of International Affairs. I must say Al—the Tribune photography does not help—surely there is some happy medium, between more hair and fewer chins.

Congratulations too, to Tony Gowron, Mike Ranosky and associates on their new Medical Centre on River Avenue.

Sorry, boys, I couldn't make that "opening" do, had to go out of town, but your whole set-up looks "deluxe"—and some day I'll drop in and pay the delayed respects. The best of luck to all including Dr. C. Benoit, who, having concluded three years post-graduate study at Mayo's, Rochester, in Opthalmology, is now associated with the River Avenue Medical Centre.

♦

July 9, 1954, St. Mary's Cathedral was the scene of the marriage of Winona Grace Livingstone and Dr. J. M. O'Keefe. Following a motor trip to New Orleans and the Southern States, I have no report!!

♦

To our Juniors, welcome and well tolerated formulae—

Dr. and Mrs. Jack D. McKenty (nee Betty White), a son, Jack Vincent, Sept. 3, 1954. What does that do to Grandpa!! Oh! well.

Dr. and Mrs. J. Gerald Fox, happily announce the arrival of Coleen Elizabeth, Aug. 13.

July 8, 1954, Dr. and Mrs. Paul Adams, Langdon, N.D., formerly of Winnipeg, announce the arrival of Paul Edward, a brother for Marie.

Aug. 10, 1954, Dr. and Mrs. A. A. Campbell (nee Pat Rutherford), welcome Peter Alexander, a new curler, etc., for Flin Flon, Manitoba.

Aug. 1, 1954, Dr. and Mrs. Rodney M. Chadwick very happily welcome a daughter in Red Deer, Alta. Hi, Rod. Congratulations.

Dr. and Mrs. Howard Dixon, 827 25th St., Santa Monica, California, announce the arrival of John Bruce, June 25, 1954.

Dr. and Mrs. H. R. Wyman, Brandon, Manitoba, on Aug. 11, 1954, welcomed Jeffrey Matthew.

Dr. and Mrs. Geo. Wortzman announced as of July 2, the birth of Ricky Marienne.

Dr. and Mrs. Duncan E. Govan, in Chicago, announce the third rugby player, or is it hockey, anyway the name is Reginald Bruce, and the birth date June 28.

Dr. and Mrs. W. Earl Shepherd announce the birth of John David, Aug. 2, 1954, in Vancouver, B.C.

Dr. and Mrs. David F. Simpson of Wilkie, Saskatchewan, announce the arrival of David Kristjon in Wilkie, Sept. 10, 1954.

Dr. and Mrs. R. H. Tavener, Cordova St., welcomed Dawna Maureen on Aug. 18.

Speaking of birthdays, many happy returns to Dr. H. D. Benwell, Grand Forks, N.D., born in Buffalo, N.Y., in 1895, graduate from Manitoba Medical—perish the thought, only a couple of years before the rest of us!

Department of Health and Public Welfare

Comparisons Communicable Diseases — Manitoba (Whites and Indians)

DISEASES	1954 Aug. 8 to Sept. 4,'54	1954 July 11 to Aug. 7,'54	1953 Aug. 9 to Sept. 5,'53	1953 July 12 to Aug. 8.'53	Total Jan. 1 to Sept. 4.'54	Total Jan. 1 to Sept. 5,'53
Anterior Poliomyelitis	31	30	853	637	101	1668
Chickenpox	42	54	33	106	1259	964
Diphtheria	0	0	0	0	0	4
Diarrhoea and Enteritis, under 1 yr.	8	15	34	29	109	150
Diphtheria Carriers	0	0	0	0	0	0
Dysentery—Amoebic	0	0	0	0	0	0
Dysentery—Bacillary	0	3	5	4	17	14
Dysentery—Bacillary Carrier	0	1	0	0	1	0
Erysipelas	1	1	4	0	20	26
Encephalitis	1	0	4	2	1	8
Influenza	6	6	10	15	64	203
Measles	73	44	25	21	827	2274
Measles—German	1	1	0	4	14	39
Meningococcal Meningitis	1	0	1	1	14	27
Mumps	31	30	32	43	919	839
Ophthalmia Neonatorum	0	0	0	0	0	0
Puerperal Fever	0	0	0	0	0	1
Scarlet Fever	7	24	10	16	399	291
Septic Sore Throat	4	1	3	49	44	76
Smallpox	0	0	0	0	0	0
Tetanus	0	2	1	0	2	2
Trachoma	0	0	0	0	0	0
Tuberculosis	87	73	48	48	452	702
Typhoid Fever	0	0	0	0	3	0
Typhoid Paratyphoid	0	0	0	0	0	0
Typhoid Carriers	0	0	0	0	0	0
Undulant Fever	1	1	0	0	5	9
Whooping Cough	21	3	22	19	66	145
Gonorrhoea	113	123	130	103	921	822
Syphilis	11	7	4	6	75	60
Infectious Jaundice	10	15	29	7	253	237
Tularemia	0	0	0	0	1	2

Four-Week Period July 11th to August 7th, 1954

DISEASES (White Cases Only)	*809,000 Manitoba	*861,000 Saskatchewan	*3,825,000 Ontario	2,952,000 Minnesota
*Approximate population.				
Anterior Poliomyelitis	30	10	40	85
Chickenpox	54	44	330	....
Diarrhoea and Enteritis, under 1 yr.	15	1	....	....
Diphtheria	....	....	3	2
Diphtheria Carriers	....	....	....	....
Dysentery—Amoebic	....	....	....	2
Bacillary	3	1	17	6
Bacillary Carriers	1	....	....	....
Encephalitis Epidemica	....	1	1	1
Erysipelas	1	1	3	....
Influenza	6	1	17	3
Infectious Jaundice	15	52	63	204
Measles	44	64	470	149
German Measles	1	41	54	....
Meningitis Meningococcus	....	....	6	5
Mumps	30	44	237	....
Ophthal. Neonat.	....	....	....	....
Puerperal Fever	....	....	....	....
Scarlet Fever	24	6	95	5
Septic Sore Throat	1	5	4	59
Smallpox	....	....	....	....
Tetanus	2	....	....	....
Trachoma	....	....	....	....
Trichinosis	....	....	....	1
Tuberculosis	73	26	76	143
Tularemia	....	....	....	....
Typhoid Fever	....	1	....	1
Typh. Para-Typhoid	....	....	....	....
Typhoid Carriers	....	....	....	....
Undulant Fever	1	....	2	13
Whooping Cough	3	4	233	106
Gonorrhoea	123	....	202	....
Syphilis	7	....	75	....

DEATHS FROM REPORTABLE DISEASES. JULY, 1954

Urban—Cancer, 64; Influenza, 1; Pneumonia, Lobar (490), 1; Pneumonia (other forms), 10; Tuberculosis, 1; Food Poisoning, 1; Septicaemia and Pyaemia, 2; Diarrhoea and Enteritis, 1. Other deaths under 1 year, 26. Other deaths over 1 year, 176. Stillbirths, 18. Total, 220.

Rural—Cancer, 31; Pneumonia, Lobar (490), 1; Tuberculosis, 1. Other deaths under 1 year, 11. Other deaths over 1 year, 144. Stillbirths, 13. Total, 168.

Indians—Pneumonia (other forms), 4. Other deaths under 1 year, 2. Other deaths over 1 year, 3. Stillbirths, 1. Total, 6.

♦

Poliomyelitis definitely is not epidemic in Manitoba in 1954. One hundred and one cases to date is quite low for a post epidemic year. Only two cases have died.

Jaundice Infectious is quite prevalent, not only in Manitoba but throughout Canada and the United States.

Tuperculosis is apparently showing a marked decline in reporting of new cases. However, these may not be true figures as there is always a lag in reporting tuberculosis.

Venereal Diseases are showing a slight increase to date in 1954. Our efforts to prevent and control these diseases have not been decreased, in fact, we are intensifying our work in this regard.

Association Page

Reported by M. T. Macfarland, M.D.

Nominating Committee Report

President—Dr. R. W. Whetter, Steinbach.
First Vice-President—Dr. R. Lyons, Winnipeg.
Second Vice-President—Dr. G. H. Hamlin, Portage la Prairie.
Dr. J. E. Hudson, Hamiota.
Honorary Secretary—Dr. H. W. C. North, Carman.
Honorary Treasurer—Dr. Jack McKenty, Winnipeg.
Rural Member at Large (three years)—
Dr. R. H. Harris, Virden.
Dr. M. Potoski, Dauphin.
Winnipeg Member at Large (three years)—
Dr. A. B. Houston.
Dr. R. A. Macpherson.

Article 11 of the Constitution and By-laws reads as follows:

"The President, First and Second Vice-Presidents, Honorary Secretary, and Honorary Treasurer, and the additional members of the Executive Committee, unless otherwise provided in this Constitution, shall be elected at the business session of each Annual Meeting. They shall be elected from nominations, one or more for each office, to be submitted by the Nominating Committee to the Executive Committee and published in the Association Review at least one month before the Annual Meeting, and from such other nominations as may be made from the floor at the business session of the Annual Meeting."

Panel Discussion

"How Should a Doctor Plan His Retirement and Estate?"

An innovation is planned for the Annual Meeting this year when a two-hour panel of well-informed persons will discuss the above topic, and answer questions which may be submitted in advance or from the floor. Wives are invited since they have a major role in planning for the future. Light refreshments will be served at the conclusion of the panel.

Participants will be: Dr. R. W. Richardson, Chairman, Committee on Economics, Canadian Medical Association; Robert K. Berry, Trust Officer; Arthur Johnston, Chartered Life Underwriter; S. B. Phipps, Investment Counsellor; Don A. Thompson, Barrister.

These men are well informed and will be pleased to give you their advice in solving your specific problems. There will be opportunity given for questions from the floor following the discussion period, however, it is recommended that you send in questions by mail now to Dr. M. T. Macfarland, Executive Secretary, Manitoba Medical Association, 604 Medical Arts Building, Winnipeg.

Hobby Show

Some of our sister divisions have introduced a Hobby Exhibit at which members of the profession present samples of hobbycraft in which they have a continuing interest. Such an exhibit will be held in connection with the Annual Meeting, October 12-14.

Dr. Alex M. Goodwin has generously consented to act as Chairman of the Committee and will be pleased to receive suggestions and volunteers. Applications to exhibit in the Hobby Show should be forwarded to the Association Office, 604 Medical Arts Building, and should include name, address and subject of exhibit.

Book Reviews

The **Book of Health** is not a doctor book in the ordinary sense. It does not tell the reader "what to do till the dotcor comes" or how every man can become his own physician. It is therefore worthy of our attention.

It is a large, copiously illustrated volume of 936 pages. The text is clearly and simply written by a corps of "medical writers" who are not doctors but who are qualified by experience to make medical topics interesting and understandable to lay readers. Over two hundred and forty medical authorities edited the matter so written in order to ensure its accuracy. It is a good arrangement. The writers make the text readable, the editors make it accurate and the illustrators make comprehension easy.

The Biblical "In the beginning" might well have opened this book also, for the first chapter is titled "Life Begins," and the first section is headed "How It Begins." From conception we are led through the seven ages of man; in a word, if not from the womb to the tomb, at least from the womb to the death-bed.

There is a chapter on "The Body, how it functions as a machine" which is a brief review of physiology; and another chapter in which the reader is introduced to the various organisms that, as it were, throw monkey-wrenches into the machine. The various systems are then dealt with seriatim, each being introduced by an anatomico-physiological section entitled "What it is and what it does," after which come little or longish articles on the ailments common to that system. The mind gets a chapter to itself and so does Geriatrics. Evidence of the completeness of the book is a chapter on "Medical History," one on "Medicine and the Law" and many useful Tables.

There is very much more to the book than what has been mentioned above. In its scope it lives up to its sub-title A Medical Encyclopedia. Probably most of the copies sold will be purchased by curious laymen but it is not by them that it will be found most useful. The people who will appreciate it most and to whom it will be of greatest value are those whose work lies in the care of the sick and on the fringes of medical practice. Thus it is an excellent book for practical nurses, a good book for pupil nurses and a useful book for social workers and others in like employment. For these its purchase would be a justifiable expense, and it should be in the library of every school where such workers are taught.

The illustrations are not only abundant (1400) but excellent and many are in colour. The index is complete and serves also as a little medical dictionary.

The Book of Health (the work of over 300 doctors, writers, artists, and photographers); edited by Randolph Lee Clark, Director and Surgeon-in-Chief, University of Texas, and Russell W. Cumley, Professor of Medical Journalism, University of Texas. The Elsevier Press, Houston. In Canada: Burns and MacEachern, 12 Grenville St., Toronto 2, Ontario. $12.50.

"Lives of Great Men . . . "

"Medical Biographies" is a series of brief sketches which deal chiefly with the ailments of thirty-three famous men. The lives of great men all remind us that greatness does not confer immunity from disease and also that "to every man, both young and old, death cometh soon or late."

There is usually a close association between a person's ailments and achievements. The lunatic, "the lover and the poet are of imagination all compact." Could Poe have written as he did had he not been a little mad? Could Keats have sung so sweetly had he not felt that Death stood close to him?

We get a better understanding of a man's works, of his successes and failures when we have a knowledge of his medical history. And to the modern medical reader of a great man's biography there is always the realization of what a doctor with such knowledge as we now possess could have altered the course of history.

These little stories are interesting and are told without bias. For example the author is not completely certain that Henry VIII was luetic as so many authors claim. The subjects of these sketches are Buddha, Charlemagne, William the Conqueror, Christopher Columbus, Henry VIII, Benvenuto Cellini, Philip II, Samuel Pepys, Sir Isaac Newton, Dean Swift, Peter the Great, Frederick the Great, Kant, Catherine the Great, Washington, Gibbon, George III, Marat, Napoleon, Byron, Keats, Poe, Darwin, Whitman, Frederick III, Garfield, Cleveland, McKinley, de Maupassant, R. L. Stevenson.

In a sense these are medical and surgical clinics with presentation of symptoms and discussion of diagnosis. They are quite readable and as accurate as the authorities from whom the data have been gathered. There are some mistakes (such as designating Henry VIII as "King of Britain" and misquoting Osler in the matter of the "Captain of the Men of Death!") But these do not detract from the interest that the book arouses or the information that it gives.

There are 259 pages and 32 full page portraits. The list of authorities quoted or consulted occupies fifteen pages. This is a very useful bibliography because it directs the curious reader to volumes which he may find it profitable to read.

Every one interested in the medicine of history will enjoy this book which sheds a light into a number of the dark corners which historians and biographers tend to leave unexplored.

Medical Biographies, the Ailments of Thirty-three Famous Persons by Philip Marshall Dale, M.D., University of Oklahoma Press. In Canada: Burns and McEachern, 12 Grenville Street, Toronto 2.

Fracture Treatment Made Clear

The author says "This review is intended to illustrate and discuss, briefly and systematically, the principal features concerning the diagnosis and treatment of fractures. It has been written for the medical student, the house officer, and the general practitioner. There has been no attempt to make the book comprehensive or complete in all details, nor has it been written to promote discussion. It is to serve instead as a supplement to the standard texts in the field."

There are three introductory chapters: 1. Anatomy and Physiology; 2. Clinical Examination of Fractures; 3. Principles of Treatment of Fractures. Then follow fourteen chapters each devoted to one region (Head, Spine, Thorax, Shoulders, Arm, Elbow, Forearm, Wrist, Hand, Pelvis, Hip and Thigh, Knee, Leg, Ankle, Foot).

Each fracture is discussed from the standpoints of etiology, incidence, pathology, types, clinical findings, complications, treatment, conservative and operative prognosis. Everything capable of illustration is illustrated. Line drawings show how the fracture can be caused and show the effects on the bone. Clear pictures show the X-ray findings. Treatment is also illustrated. No book could put the subjects more clearly. The text is brief. Statements are positive, precise and clear.

Including glossary and index there are 228 pages and 605 illustrations.

Illustrative Review of Fracture Treatment, by Frederick Lee Liebolt, A.B., M.D., Sc.D., LL.D. Surgeon in charge of Orthopedics, the New York Hospital, etc. Lange Medical Publications, Los Altos, California, 1954. Price $4.00.